Functional Diagnosis

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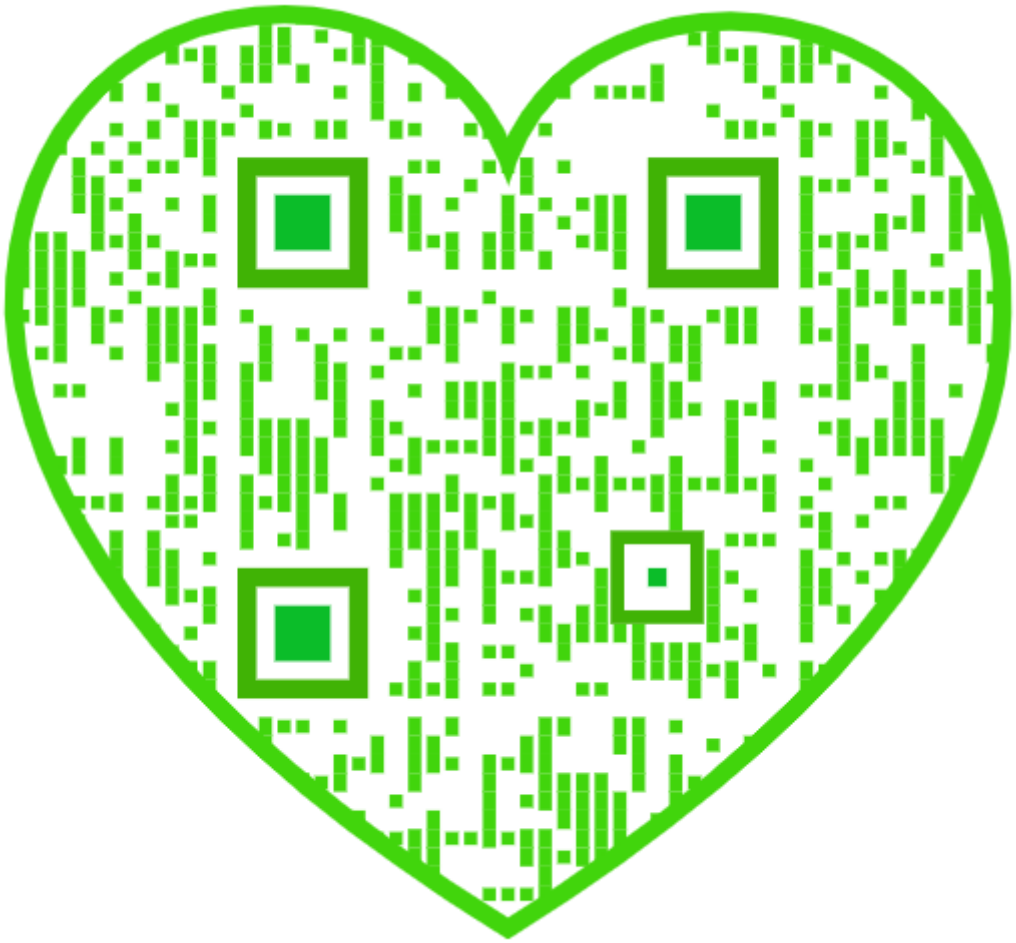

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FUNCTIONAL DIAGNOSIS

THE

Application of Physiology to Diagnosis

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INTRODUCTION.

Two considerations have led to the compilation of this work.

1. The author's experience as a teacher of physiology to first and second year students, and of neurology to third and fourth year men, has forced upon him the conclusion that, no matter how thoroughly the former subject may be instilled into the student during the early years of his course, or how intelligently received by him, the teacher of diagnosis in the latter years is always obliged to retrace a great deal of the ground in order to elucidate the relations betewen the normal and the abnormal, and above all, the working value of symptoms in interpreting functional perversion.

The accomplishment of this retracement with the maximum directness and economy, it seemed to the writer, would be greatly facilitated by the use of a text book which should set forth these relations in a sequential and systematic form—a thing which no text book on diagnosis has thus far attempted Or possibly a yet more excellent way would be to use this book in teaching freshmen and sophomores physiology, so that by the time they were ready to take up diagnosis they would already have a basic understanding of the sequential relations between normal and deranged function, as manifested in the symptoms of various diseases.

2. In the maturer practice and literature of diag-

nosis precisely the obverse of the above condition prevails. There is an obvious hiatus in the current teaching and practice of the subject—a missing link, so to speak, between physiology and pathology. Current text books base their diagnostics upon the isolated, objective, morbid conditions which constitute the disease-complex, and which too often represent the last structural changes wrought by disease, rather than the functional derangement in which disease usually has its beginnings. The spirit of the times calls for a prophylactic type of diagnosis—one that shall detect disease in its functional outposts.

It is of prime importance that the diagnostician be able to reason back his symptoms to their functional premises, and to state his pathologic condition in terms of physiology. It is of equal importance that he be able to establish this functional relation as directly and as close to the normal as possible, so that his diagnosis may be as nearly as possible a proximate principle. This is where, in the author's judgment, all of the many excellent works on diagnosis now in the field are lacking, and it is in the hope of making good this lack, and establishing a direct, sequential connection between normal physiology and disease symptoms, that this work on functional diagnosis is offered. It attempts the task of translating the normal functions of the body, through their various derangements, into terms of disease.

Those parts of the book which treat of pure physiology are so arranged and printed as to constitute in themselves a fairly complete text book on that subject, though necessarily condensed. The avowed purpose of the work being to point out the functional significance of symptoms, it deals purely with function and not at all with anatomy. The reader is presumed to be already acquainted with anatomy, or must gain his acquaintance from another book.

No attempt is made to elaborate, far less to argue, the pros and cons of those disputed theories which still divide the ranks of physiologists. Where a decision is necessary, the author has carefully canvassed the concensus of opinion, as reflected in recent literature, and has boldly adopted the theory which commands the preponderance of evidence.

A consideration of the optics of the eye and the acoustics of the ear has purposely been omitted. In their pure intent, these phases of vision and hearing have no bearing upon pathology, and are therefore not germane to a study of functional diagnosis. Convergence and accommodation, being integral parts of the physiology of vision, are dealt with.

Naturally the function of reproduction, in its strict interpretation, does not lend itself very extensively to pathologic application. The only pathological question in this connection, having a strictly physiologic value, is "to reproduce or not to reproduce?" The other phases of the subject belong

to a consideration of embryology and obstetrics. However, the point has been strained to the extent of considering the commoner disturbances of menstruation, and the elementary aspects of abnormal parturition, such as muscular inertia, and the like. No very detailed application has been attempted in this direction. The line had to be drawn somewhere, or it would have necessitated a text book on gynecology and obstetrics.

The author hopes that his efforts will be found helpful to both student and practitioner in establishing a sequential link between physiology and morbid function, and enabling him to make an intelligent functional interpretation of the symptom-complex of disease.

NOVEL ADVANTAGES OF THE WORK.

It presents—as no other book on diagnosis does—the normal function, and the derangement of it which constitutes disease, in sequential relation. Every normal function of the body and its organs is briefly but clearly described, its mechanism and significance explained; immediately following are given the various derangements to which, in the nature of them, these functions are subject; the symptoms to which, because of their influence upon the normal function these derangements give rise; the diseases in which these derangements occur, and why.

This is an entirely new and much-needed standpoint of diagnosis, and offers the following valuable practical features:

1. It bridges the gap, left by current works on diagnosis, between normal function and the symptoms of disease. The mere symptoms themselves, detached from the normal of which they are a perverted index, are made the ordinary basis of diagnosis.

2. It enhances the functional element in diagnosis, giving it a dynamic rather than a static character, regarding the body as a going machine, whose diseases are aberrations of function rather than structural changes.

3. It enables the student and practitioner to reason back the pathologic findings to their functional

premises, and thus to gain an intelligent conception of the fundamental trouble, irrespective of the name-classification of the disease.

4. It furnishes a functional, rather than a pathological classification of diagnostic data, by which symptoms may be interpreted according to their physiological import.

(For example, a derangement of respiration is often not directly due to a defect in the respiratory organs, and its explanation cannot therefore be found under Diseases of the Respiratory System, as ordinarily classified. In this bok all derangements of the respiration are expounded under Respiration, because respiration is treated as a function, and everything that influences or modifies it is included under that heading.

5. It furnishes a combined text book on physiology and diagnosis—two subjects which are fundamentally related—a distinct advantage to the student of these subjects, and no less advantageous to the physician who practices diagnosis.

FUNCTIONAL DIAGNOSIS.

The ascendancy of the micro-organism and the marvelous advances in laboratory methods have of late years lent an exaggerated importance to these agencies of diagnosis, to the discounting of the functional element. Symptomatology has almost wholly given place to objective and especially to laboratory findings. Further than this, there has been a growing tendency toward scientific specialism, and while physiology as an independent science has of late made gigantic strides, and greatly enlarged the scope of its jurisdiction, there has been an equally growing tendency to divorce it from its mutual relations with other branches of medicine which have undergone similar expansion.

It is in the hope of contributing, however feebly, to the re-establishment of these neglected relationships upon a practical clinical footing that this work on applied physiology is offered. Disease is, after all, essentially a phenomenon of function, of which structural aberrations are but the resulting manifestations. As Dr. Putnam said, in his masterly address before the Congress of Arts and Sciences: "No anatomical research can pierce the secret of broken co-ordinations, and yet it is in these that a great part of disease begins, or eventually comes to consist." It is therefore of prime importance that the diagnostician be able to reason back his symptomalogical data to their functional premises, and to

state his pathological condition in terms of physiology. And it is with a view of assisting him in the process that the writer has here attempted the inverse task of translating physiological phenomena, through functional aberration, into terms of pathology.

RESPIRATION.

The Lungs may be considered as two large sacs which communicate with the external atmosphere by means of the trachea, the external walls of the sac being in adherent relation to the walls of the thorax. The thorax, outside of the lung-sac, is completely shut off from communication with the atmosphere and with the abdomen.

Retractions.—Owing to the adherence of the lung sac to the chest walls, a cicatrix in the former will pull in the external covering of the chest and produce a depression. These depressions are often seen in tubercular cases where there has been an old cavity that has cicatricized. They may be found in any of the intercostal spaces, but are most common in the clavicular and subclavicular spaces, due to apical cicatrices.

Pneumothorax.—In cases of tuberculosis and other processes of lung degeneration the lung sac frequently ruptures under exertion or cough, and air is forced into the thorax at each inspiration. This condition is known as **pneumothorax.** It may, of course, also result from a penetrating wound of the chest wall.

Respiratory Sounds.—The two types of sound heard in normal respiration are (1) that produced by the passage of the air through the bronchi, called bronchial breathing, and (2) that produced by the

inflation of the lung alveoli, called vesicular breathing.

Bronchial breathing sounds, as might be expected, are rough and sonorous, and are equally prominent in inspiration and expiration, except for the different force with which the air is driven through the tubes.

Vescicular breathing sounds are softer and more blowing in character; and inasmuch as they are caused by alveolar inflation, they are confined almost entirely to inspiration, the almost sudden recoil of the lungs in expiration putting a rapid end to the vescicular sound.

Normal breathing sounds heard through the chest wall are made up of a combination of these two elements. The bronchi being at almost every point overlaid with lung (always containing more or less air) the bronchial sounds are not well conducted to the chest wall, and are therefore in normal breathing greatly subordinated to the vescicular sounds. This is especially the case in inspiration, when the alveoli are filling with air. In expiration, when the alveoli are recoiling and emptying, the bronchial sounds become more dominant.

In infants and young children, in whom the lung tissue is relatively undeveloped, the bronchial sounds are normally much more dominant than in adults.

Bronchial Breathing.—Anything which lessens the layer of air-containing lung between the bronchi

and the chest wall, or increases the conductivity of the medium, exaggerates the bronchial element in the respiratory sounds, and gives what is known as bronchial breathing. Chief among these influences, of course, is **consolidation of lung tissue**, which has both the effects enumerated above.

Bronchial breathing is therefore found in **pneumonia, tuberculosis, syphilis of the lung**, etc., over the areas consolidated by the process.

Vescicular Breathing is found in just the reverse premises to those which cause bronchial sounds, i. e., in those which increase the amount of air-containing lung between the bronchi and the chest wall. Such a type of breathing is heard in **emphysematous** conditions, especially vicarious and surgical emphysema, and **asthma.**

Stridulous Breathing, a harsh whistling sound during inspiration, is frequently heard in cases of tracheal and laryngeal obstruction by **spasm, oedema, membranous croup, severe catarrhal inflammation**, etc., the sound being due to stenosis of the air passage.

Hiccough.—Sometimes the glottis closes spasmodically before inspiration is completed, producing a sudden stenosis of the air passage and thus causing a spasmodic stridor. This is familiarly known as hiccough.

Vescicular Breathing is exaggerated by any conditions which increase the amount of air lying between the bronchi and the chest wall, as, for instance,

emphysema, before the elasticity of the lining is impaired, especially **vicarious emphysema.** Vesicular sounds are diminished without any special exaggeration of bronchial sounds, (1) by any conditions which impair lung elasticity, as **emphysema** in later stages, **oedema,** etc., (2) by the presence of air or water in the surrounding thorax (**hydro** or **pneumothorax**), owing to pressure exerted on the lung; (3) by **pleuritic pains** limiting the movements of the chest; (4) by occlusion of air passages, lessening the entrance of air into the lungs.

In **bronchitis** the breathing is of a bronchial type in the early stages, because of the exaggerated roughness of the tubes; later, it becomes vescicular, owing to the emphysema and loss of elasticity of the lungs.

Suppressed Breathing Sounds.—Where the lungs are surrounded with air or water, as in **pneumo** or **hydro-thorax,** both tubular and vesicular sounds are muffled and in many cases almost inaudible.

PERCUSSION.

The principles of percussion are based upon anatomical, rather than upon physiological, premises. In general, percussion sounds may be said to depend upon the anatomical conditions corresponding to the physiological conditions accounting for auscultatory sounds. Generally speaking, in instances and locations where the bronchial sounds predominate, as in consolidations of **pneumonia, tuberculosis, syphilis, gangrene,** etc., the percussion note is dull or flat; where the vesicular sounds are in the ascendant, percussion gives a hyper-resonant note, as in **emphysema, asthma,** etc.

However, as the pitch and quality of the percussion note depend upon the relative resistance of the part percussed rather than upon its conductivity, there are exceptions to this correspondence between auscultation and percussion. For example, a cavity in the lung will give bronchial sounds, because the vesicular tissue is gone from between the bronchi and the chest wall, but percussion over a cavity will yield hyper-resonance. **Pneumothorax** and **hydrothorax** alike diminish both vesicular and bronchial sounds, but the former gives a hyper-resonant note because of its elasticity, upon percussion and the latter a flat note because of its solid resistance.

INSPIRATION AND EXPIRATION.

Inspiration is accomplished by enlarging the capacity of the thorax by means of the muscles; the lungs, being adherent to the walls of the thorax, are expanded and air rushes in by way of the glottis and trachea to fill the vacuum thus created. Inspiration is therefore an active muscular process.

Expiration is accomplished by the elastic recoil of the lung and the weight of the ribs—purely physical forces—and is a passive return to equilibrium.

Delayed Expiration.—Any condition which interferes with the free expiration of air from the lungs exerts increased resistance to the recoil of the elastic tissues and to the gravity of the ribs, and hence prolongs the passive process of expiration. **Bronchitis, asthma, nasal** and **laryngeal catarrh, enlarged tonsils** and **growths,** all of which block the air passages, are prominent illustrations. The same result attends any pathological lesion which impairs the elasticity of the lungs. Delayed expiration, if long continued, eventually results in undue retention of air in the lungs (**Emphysema.** See below), and the external circumference of the thorax is enlarged (**barrel chest**).

Prolonged Inspiration is seen in any condition which interferes with the free passage of air into the lungs through the glottis and trachea. As the same causes, however, usually interfere equally with

expiration, the relative lengths of the functions is the same as those described under delayed expiration. **Movable tumors of the larynx** occasionally interfere with inspiration and not with expiration.

Forced Inspiration.—Under any pathological condition which renders the performance of inspiration difficult, or which necessitates a greater number of inspirations in a given time, the ordinary muscles of inspiration have to be reinforced by the accessory muscles of respiration, namely, the sternocleidomastoid and those muscles which control the size of the nasal and tracheal openings. The involvement of the muscles in the act of inspiration will be found an unfailing indication of seriously embarrassed respiration, such as occurs in **pneumonia, emphysema, pulmonary oedema, advanced tuberculosis,** all those diseases of the heart and thorax which impede inspiration (**hydro** and **pneumothorax**), and all conditions which obstruct the air passages, as **laryngeal oedema, growths, membranous croup, laryngismus,** etc.

* * *

Mechanism of Inspiration.—The enlargement of the thorax takes place in its cross diameters by the raising of the ribs and in its vertical diameter by the lowering of the diaphragm by contraction.

In regard to the first process, it may be said that all those muscles whose contraction elevates the ribs are muscles of inspiration. However, all such muscles are not haibtually used in normal inspiration.

Those which ordinarily take part in the process are the costals, intercostals, scaleni and serrati.

It must be remembered that the ribs normally slant downward from the spine ventrally, so that when their ventral ends are raised the distance between the spine and the sternum is increased. In like manner, the line of junction between the rib and the spinal tubercle is inclined downward laterally, so that elevation of the rib increases the lateral diameter of the chest.

The second process, the descent of the diaphragm is accomplished by the drawing downward of the central tendon. The downward pressure of the diaphragm on the abdominal organs causes them to bulge the abdomen outward. Diaphragmatic breathing is, therefore, called abdominal breathing.

The lungs, being adherent on all sides to the chest wall, expand in every direction that the chest enlarges, and fill with air. The muscles, therefore, have to overcome (a) the weight of the ribs and (b) the elastic pull of the lungs.

Unequal Expansion of the two sides of the thorax may be due to an impairment of the elasticity of the lung tissue on one side or to an immobility due to the binding down of the pleural sac by **old adhesions** (in which case the outer wall will be retracted as already described), or to **effusions** or **tumors in the thorax** (in which case the immobile side will be distended).

Litten's Phenomenon.—In the position of respiratory rest, the diaphragm lies flat against the thorax from its attachment to about the sixth rib. Upon inspiration this muscular layer "peels off," as Cabot says, "as it descends and allows the lung to take its place." This "peeling off" of the thick diaphragm and its replacement by the line of lighter lung tissue can be seen, in a proper position and light, as a shadow moving down the lower part of the chest wall.

In order to perceive this phenomenon, the patient must lie flat on his back, his entire chest bared, his feet turned toward the only source of light in the room, the observer seated at his side, and must take a full breath. The shadow, of course, depends upon the ripple of different level that passes over the chest surface as the thick diaphragm recedes and gives place to the thin lung tissue.

In **pleuritic adhesions**, the movements of the diaphragm are prevented; in **effusions, emphysema** and **pneumonia**, the diaphragm is separated from the chest wall, hence the shadow is not seen. Solids or fluids under the diaphragm, unless extremely large, do not interfere with its movements.

TYPES OF BREATHING.

The natural type of breathing is that which starts with the diaphragm, the first perceptible movement being an obtrusion of the abdomen. Thence the process spreads upward, involving the ribs and shoulders. This type is called the abdomino-costal type, and is almost universal among men. In civilized women, on the contrary, the diaphragm takes but little part in respiration and the ribs in consequence acquire an exaggerated importance; this is called costal breathing. Investigation demonstrates that it is due to a difference of dress, and not to genuine sexual differences. The types may be, and often are, modified by disease.

Pathological Variations of Type are seen in certain diseases. In diseases of the chest, where costal expansion causes pain, as in **pleurisy, pneumonia,** etc., and where intrapulmonary pressure is increased, as in **hydro** or **pneumo-thorax** and **emphysema,** the diaphragm is the main factor in respiration, producing the abdominal type of breathing.

In abdominal lesions, on the contrary, such as **tumors, ascites** or **visceral enlargement** impeding the movements of the diaphragm, the breathing is mostly costal.

In **paralysis of the phrenic nerves** the breathing is, of course, costal, and in **paralysis of the intercostals** the reverse. The latter is, however, rare except immediately preceding death.

Reflex Inspiration.—We have already discussed the question of the automaticity of the respiratory centre. But whether it is normally an automatic center or not, it is undoubtedly a reflex one, in direct connection with and subject to reflex stimulation by most, if not all, of the spinal and cranial sensory nerves. This is familiarly illustrated by the muscle-sense, etc. That it is also subject to reflex stimulation from the cerebrum is shown by the effects of emotion upon breathing.

Naturally, however, one would expect the sensory nerves of the respiratory tract to exert the most powerful and constant reflex influence upon the centre, and this is the fact. The branch of the vagus nerve especially, which is distributed to the lung itself, furnishes very important sensory reflex stimulation. Experiment makes it probable that this nerve contains both inhibitory and accelerator fibres, regulating the rhythm of the lungs in much the same way that the inhibitory and accelerator nerves regulate the rhythm of the heart. Peripheral stimulation for this reflex regulation is, of course, furnished by opposite conditions of the lung, collapse calling into play the accelerator and distension the inhibitory fibres.

Rapid, Shallow Breathing.—The above is undoubtedly one of the explanations of rapid breathing in pathological conditions where only shallow inspirations are possible, as in **pneumonia,** from consolidation of lung tissue, **atelectosis,** from alveo-

lar collapse, **pleurisy,** from the pain caused by inspiration, **hydro** and **pneumo-thorax,** from compression, and all diseases of the heart which exert a backward pulmonary pressure. Of course a further explanation is found in the relation of blood oxidation to circulation (see below).

Pain in the Chest.—As the lungs themselves are supplied with no nerve fibres of pain, any thoracic pain connected with respiration and referred to as "pain in the lungs" must have its seat in one or other of the serous sacs surrounding the lungs (the pleurae) or in the bronchi. Hence, when any pulmonary disease is accompanied by pain on respiration, it is due to an attendant **bronchitis** or **pleurisy.**

Asthma is a conspicuous illustration of perverted reflex influence upon respiration. Asthma is the result of spasm of the bronchi, and may be excited by nasal obstructions and abnormalities, irritations of fauces and larynx, indigestion and other peripheral stimuli, acting through the respiratory centre in the medulla.

Asthma of Renal Diseases is produced by the toxic influence of the blood upon the centre of respiration direct.

Expiratory Centre.—In normal respiration, inspiration is the only active factor, expiration being a purely passive physical process, needing no innervation. Hence the automatic centre of respiration must be regarded as an inspiratory centre only.

However, under certain conditions active expiration does take place, and is always rhythmical and co-ordinated, so that we must suppose it is controlled by a centre; and as after section of the medulla, no expiratory movement has ever been demonstrated, it is reasonable to locate the centre in the medulla. Wherever it is located, it is not normally automatic; and, as Howell points out, "its activity must be dependent in some way upon that of the inspiratory centre, as even our most violent respiratory movements show an orderly sequence of inspiration and expiration." This author suggests that "the action of the expiratory centre is conditioned upon the previous discharge of the inspiratory centre, just as in the heart beat the contraction of the ventricle is conditioned upon the previous systole of the auricle." The stimulation of the expiratory centre may, in general, be said to consist in exaggerations of the stimuli which normally excite the inspiratory centre.

Automatic Stimulus.—Automacy is, of course, only another term for "constant reflex produced by a stimulus originating within the organism." The automatic action of the respiratory centre depends upon such a stimulus. We should naturally expect to find the source of the stimulus in the function which the centre controls, namely, the interchange of the gases in the blood, and experiment has demonstrated that it consists chiefly in the accumulation of carbon dioxid in the blood, although

the diminution of oxygen also undoubtedly plays a part.

The reflex is set going in the newborn child doubtless by the severance of placental circulation and the consequent increase in the carbon dioxid of the blood. Possibly the stimulus of the air and tactile influences to the skin help.

Haldane and Priestley (Journal of Physiology, xxxii. p. 225) advance what seems to be definite proof that the activity of the respiratory centre is normally governed by the pressure of carbon dioxide in the alveolar air and consequently in the arterial blood. The experiments consist of careful analyses under a variety of conditions of (a) samples of air at the end of a quick, deep expiration, following inspiration; and (b) samples of air at the end of a deep expiration at the usual time, the average of (a) and (b) being taken as the average alveolar air. Making these tests in conditions of bodily rest (1) at ordinary atmospheric pressure, (2) on top of a mountain, (3) at the bottom of a mine, and (4) in a pressure-chamber they found that the percentage of CO_2 of the alveolar air reduced to a pressure of one atmosphere and a temperature of zero degrees was a constant; the oxygen, however, varied all the way from 10.4 to 26.8 per cent. The ventilation in these cases is to be considered constant. In case of a voluntary increase of frequency of respiration the percentage of CO_2 in the alveolar air remains constant; that is, the increase

in frequency is balanced by decrease in depth. If the percentage of CO_2 in the inspired air was varied, it was found that so small an increase as .2 per cent in the CO_2 of the alveolar air doubled the ventilation of the lungs. Under such conditions the oxygen of the alveolar air was always above normal. On doing work the percentage of CO_2 in the alveolar air is found to be increased. In apnoea the CO_2 of the alveolar air was diminished.

Zuntz and Geppert found that the arterial blood, in case of work, contained less CO_2 than when no work was done, and therefore they thought that CO_2 was not the cause of the hyperpnoea of work. But Haldane and Priestley state that while the total CO_2 is diminished in the arterial blood during work because of increased acid production, the pressure of free CO_2 on the contrary is increased. They thus dispose of the supposition that some other product of muscular action is the cause of work hyperpnoea and make it probable that CO_2 is the active agent in the stimulation of the respiratory centre.

Rapid Breathing in Fevers.—Doubtless the increased frequency of breathing in fevers, especially infectious and low grade fevers, where the metabolism is rapid, is due in some measure to the accumulation of CO_2 in the blood, acting as an abnormally powerful stimulus to the respiratory centre. It would seem, however, that this centre, like most others, can be over-stimulated, for CO_2 narcosis in

such fevers slows and enfeebles the respirations and finally paralyzes them.

Impure Air.—The same remarks apply to cases of respiring air poor in oxygen and rich in CO_2.

PRESSURE CONSIDERATIONS.

Intrapulmonic and Intra-Thoracic Pressure.—Since the lungs are in communication with the external atmosphere, it follows that the air pressure in the lungs at any time of rest, whether at the end of inspiration or expiration, is equal to that of the atmosphere. When the lungs are expanded in inspiration, their capacity increases more rapidly than the air coming through the narrow glottis can keep pace with, and there is a temporary fall in pressure until the pause at the end of inspiration. Contrariwise, the recoil of the chest is a little too rapid for the outgoing air to equalize, and there is a rise in pressure. This variation, however, is in normal respiration very slight.

In the part of the thorax outside the lungs, on the other hand, since it is protected from atmospheric pressure from outside by the chest-wall, and that which reaches it through the lung-sac is modified by the elastic recoil of the lung, the pressure is always negative. The more the thorax is enlarged, stretching the lungs with it, the more forcibly this elastic recoil is brought into play and the more negative becomes the intra-thoracic pressure. It may at any given moment be represented as that of one atmosphere less the elasticity of the lungs.

Emphysema.—Any influence which interferes with the free expulsion of air through the trachea in-

creases the intrapulmonary pressure, and opposes extra resistance to the elastic recoil of the lungs, bringing into play the muscles of forced expiration to drive out the air. If this intrapulmonary pressure is kept up for any considerable length of time, the elasticity of the lungs at last becomes seriously impaired and they are no longer properly emptied. As a consequence they are in a constant state of more or less inflation which is known as **emphysema.** The same result occurs from any pathological process in the alveoli themselves, which impairs their elasticity; also physiologically in old age.

This condition is seen notably in **chronic bronchitis,** and **bronchial asthma.**

Vicarious Emphysema is seen in parts of the lungs where the other portions are temporarily or permanently incapacitated, or in one lung where the other is out of commission. The extra duty entailed upon the sound area causes an undue inflation and retention.

Cardiac Involvements.—Sooner or later, of course, the extraordinary and constant expansion of the lung causes a backward pressure upon the pulmonary artery and interrupts the cardiac cycle, mechanically and also by virtue of inadequate oxidation of the blood. This is compensated for a time by hypertrophy of the right ventricle, but eventually compensation is broken and stasis ensues.

Pneumothorax.—In **tuberculosis** and other degenerative processes the pleural sac occasionally rup-

tures under slight exertion and air is forced into the thoracic cavity at each inspiration. Or the same result is produced by a **penetrating wound of the chest,** admitting air from outside. This, of course, immediately raises the intrathoracic pressure to that of the atmosphere, and as this entrapped air does not escape as readily as it enters, the pressure is soon raised above that of the lungs, embarrassing their expansion and the heart action, and causing death if not relieved.

Coughing.—Any attempt at expiration with closure of the glottis of course raises the intrapulmonic pressure. A conspicuous and extreme example is seen in the familiar phenomenon of **coughing.** The irritation of the tubes causes a spasmodic closure of the glottis at the same time a violent effort is made at forced expiration, raising the pressure considerably, and resulting in the explosive opening of the glottis and expulsion of air under high pressure which constitutes the noisy "cough." During the period of compression pulmonary circulation is interfered with, general venous stasis occurs, and if long continued causes cyanosis, and occasionally rupture of one of the vessels.

Suction Effect of Negative Pressure.—From the foregoing it is seen that the large veins above and below the thorax are subject to atmospheric pressure; the venae cavae and the right auricle, on the contrary, are under a pressure of less than one at-

mosphere. This difference in pressure levels acts as a suction influence, assisting the flow of venous blood to the heart. It is, of course, more marked with every expansion of the lung in inspiration which makes the pressure still more negative.

In **pneumo** and **hydro-thorax**, of course, this suction phenomenon is lacking, because the intrathoracic pressure is raised to the level of, and sometimes even higher than, the intrapulmonic. This helps to explain the venous stasis occurring in these conditions.

Respiratory Waves of Blood Pressure.—Experiments show that during inspiration there is first, at the beginning of the inspiratory act, a slight fall in blood pressure, then a rise; at the beginning of expiration there is a slight rise, followed by a noticeable fall. These phenomena are explained as follows:

At the commencement of inspiration the capacity of the lung-capillaries is suddenly increased, lessening peripheral resistance and causing a momentary fall in pressure. This is quickly out-balanced by the following occurrences, both of which tend to raise the pressure: (1) Suction of increased negative pressure in the thorax, causing increased flow of blood into the heart; (2) difference of atmospheric pressure level in the pulmonary artery and vein, causing an increased flow of blood out of the heart.

At the beginning of expiration the sudden contraction of the lung-capillaries causes a momentary

rise in pressure, which is quickly overwhelmed by a reversal of the conditions described under (1) and (2) in the previous paragraph, producing a distinct fall in pressure.

Respired Air.—The air respired by the lungs is classified in various fractions of its volume according to their exchange under various physiological conditions. The classification is as follows:

Tidal Air.—The amount of air breathed in and out during a normal respiration.

Complemental Air.—The amount of air that can by forced inspiration be taken in in excess of the tidal air.

Supplemental Air.—The amount that can by forced expiration be expelled in excess of tidal air.

Residual Air.—The amount which remains in the lungs even after the supplemental air is expelled.

Minimal Air.—The small amount which is captured by the collapse of the small bronchi in an exposed and collapsed lung. A lung that has once breathed never thereafter becomes entirely airless; it always retains the minmial air even after death.

Vital Capacity is the term used to express the amount of air that a person can inspire and expire, and is, of course, equal to the tidal air plus the complemental and supplemental.

Pressure Conditions of Oxygen in Respiration.—The pressure of the atmosphere is equal to a column of mercury 760 mm. high, and is expressed as 760

mm. Hg. According to the law of partial pressure of gases, the oxygen in the atmosphere, forming one-fifth of its volume, exerts one-fifth of its pressure—i. e., 152 mm. Hg. This pressure determines the point of equilibrium at which oxygen is held in solution or dissociable combination by a liquid in the presence of atmospheric air. At this pressure, the hemoglobin of the blood is saturated with oxygen and as long as the oxygen-pressure in the atmosphere is maintained the oxygen equilibrium between the blood and the air is maintained at saturation point. If the proportion of oxygen in the atmosphere is increased the equilibrium is disturbed and oxygen flows from the air to the blood (to the limit of absorption by the plasma); if the proportion of oxygen in the atmosphere is diminished the equilibrium is disturbed the other way, and oxygen flows from the blood to the air. The same condition obtains if for the air is substituted another environment containing a more or less pressure of oxygen.

The blood at the surface of the lungs is practically under 152 mm. Hg. oxygen pressure (i. e., one-fifth atmospheric pressure.) In its course along the arteries this oxygen pressure is gradually lowered as it comes into contact with greater and greater areas of tissue devoid of oxygen. By the time it reaches the capillaries the oxygen pressure has come down to 76 mm. Hg. In its passage through the capillaries, where this low pressure area is greatly increased, the oxygen pressure is rapidly reduced, and the oxygen

just as rapidly given off to preserve the oxygen equilibrium. When it reaches the lungs again, with very little oxygen in it, it is suddenly subjected once more to an oxygen pressure of 152 mm. Hg. and saturated with the gas.

Difference of Pressure at Lungs.—Experiment shows that the relative pressures of oxygen in the alveolar air, where the exchange is made, and the venous blood that is brought to the lungs by the pulmonary arteries are as follows:

Alveolar air—83 to 129 mm.

Venous blood—40 mm.

It is evident that the exchange takes place from the air toward the blood.

Insufficient Oxidation.—It will be readily seen that any diminution in the oxygen pressure at the lungs will result in an abnormally small impartation of this gas to the venous blood, and consequently in poor oxygenation of the tissues. This may occur either from (a) poverty of the surrounding air in oxygen or (b) any condition of the respiratory mechanism which tends to rarify the air as it enters the lungs—e. g., **stenosis of the trochea or bronchi**—causing undue negative pressure during inspiration; or **emphysema,** in which the inspired air is continually diluted by an excess of residual air.

Chemical and Physical Changes in Respired Air.—Expired air differs chemically from inspired air in having lost a part of its oxygen and acquired

carbon dioxid. This is indeed the chief end of respiration, the carrying of oxygen to the blood and the removal of carbon dioxid. The proportion of gain and loss depends, of course, upon the character of the inspired air, depth of inspiration, etc. Under normal conditions, however, the exchange is about as follows:

	Insp'd.	Exp'd.	O Lost.	CO_2 Gained.
Oxygen	21	16	.05	..
Carbon dioxid....	00.4	4.5	..	.04
Nitrogen	79	79	..	..

It will be seen that more oxygen is lost than is represented by the carbon dioxid which replaces it. The excess oxygen is utilized in oxydizing hydrogen in the body to form water.

Physically the expired air differs from inspired air in being warmer (equal to body temperature) and saturated with water vapor from the lung surface. This loss of heat and water is a subsidiary function of respiration in regulating the temperature and moisture of the body.

Pressure Conditions of Carbon Dioxid.—The partial pressure of carbon dioxid in the atmosphere, on the other hand, is only four-hundredths of the atmospheric pressure, or 30.4 mm. Hg. At this pressure the blood contains 38 per cent by volume of carbon dioxid. During the passage of the blood through the arteries and capillaries, the carbon dioxid pres-

sure increases, just as the oxygen pressure decreases, and carbon dioxid flows to the blood, taking the place of the oxygen which under decreased pressure has passed to the tissues. When the blood reaches the lungs again, containing 45 per cent of carbon dioxid, it is suddenly subjected to the low CO_2 pressure of the atmosphere, and parts with enough to adjust CO_2 equilibrium with the atmosphere.

Difference of Pressure at Lungs.—The relative pressures of carbon dioxid in the alveoli and in the venous blood are as under:

Venous blood—45 mm.

Alveolar air—28 to 41 mm.

Showing an inevitable exchange in favor of the air.

Insufficient Decarbonization.—Any increase of CO_2 pressure in the alveoli will, of course, result in an insufficient diffusion of this gas from the blood.

Pressure Condition of Nitrogen.—Experiment has shown the amount of nitrogen in arterial blood and in venous blood to be the same—namely, 1.7 per cent—and as this quantity corresponds with the coefficient of absorption of nitrogen for blood at body temperature—i. e., the amount absorbed by 1 cc. at a pressure of 1 atmosphere—it may safely be concluded that nitrogen enters into merely physical solution in the blood, in accordance with Dalton's law, and plays no part in the respiratory function.

Internal Respiration.—It is, of cource, in the tissues supplied by the blood that the exchange of

gases which constitutes respiration actually takes place. In the tissues surrounding the capillaries CO_2 is being rapidly formed by metabolism, and the CO_2 pressure is therefore high. Oxygen, on the contrary, is being constantly utilized for carbo-hydrate compounds (too stable to be dissociated) and O pressure is therefore low. In the blood, as it enters the capillaries, the pressure conditions of the two gases are the reverse of this—hence the flow of oxygen to the tissues and of carbon dioxid to the blood. Both of the exchanges take place through the plasma of the blood and the surrounding lymph.

CO_2 Narcosis.—Any interference with the process of respiration, unless compensated for, will ultimately result in an accumulation of CO_2 in the tissues to an extent which will render them inert and unable to perform their proper functions. This condition is known as CO_2 Narcosis, and is the cause of death in all diseases which kill by impeding respiration.

* * *

Variations in Respiratory Frequency.—From what has gone before it is evident that the rapidity of respiration may be modified by either one of two general conditions:

(1) By any condition influencing the expansion and recoil of the lungs, acting as a stimulant of the accelerator or depressor nerves of the lungs.

(2) By any condition influencing the quantity of

carbon dioxid in the blood, acting as a direct stimulant of the respiratory centre.

Physiological variations are probably rarely, if ever, primarily due to the first of these groups of conditions, as it is hard to conceive of any set of normal conditions which could directly cause an exaggerated or diminished expansion of the chest. The pathological conditions which influence this feature of respiration are described elsewhere.

The latter group includes (a) varying degrees of metabolism and consequent variations in carbon dioxid production, (b) conditions influencing the gaseous exchange in the lungs or tissues, and thus influencing the quantity of CO_2 in the blood, (c) the velocity and pressure of the blood, and its oxygen-carrying power. These influences may all be, and continually are, exerted in varying degree in the ordinary course of physiological activity, causing continual variations in the frequency of respiration, which are familiar to everyone.

The increased frequency of breathing during muscular exercise, the diminished frequency during rest and sleep, the increased rate under vitiated atmospheric conditions and decreased rate under pure air, the differing effects of heat and cold, hunger and satiety, are well-known phenomena. Of these, the influences of metabolism, and especially muscular exercise, where CO_2 is very rapidly produced, are the most common.

It is a well-known fact that the respiration is

relatively rapid in infancy, 35 to 40 per minute, gradually diminishing in frequency until in adults it remains pretty constant at 18 to 24 per minute. This is doubtless due in large measure to the relatively smaller available lung capacity in children, which increases in greater ratio than the growth of the body; and to the relatively greater metabolic activity of the tissues in childhood consequent upon growth.

Fevers.—Undoubtedly the increased freqneucy of respiration in fevers, as already explained, is largely due to accumulation of carbon dioxid.

Pleurisy causes rapid respiration because the limited expansion of the time chest due to the pain sets in motion the reflex mechanism of the vagus accelerators.

Pneumonia, phthisis, etc., exhibit rapid breathing because of the small amount of available lung area, operating both by way of the vagus and by the accumulation of CO_2 in the blood.

In **hydro** and **pneumothorax** the movements of the lungs are limited by the pressure in the thorax; here both the vagus reflex and the excess CO_2 in the blood operate to increase the frequency of respiration.

In **emphysema** and **atelectasis** the excess CO_2 appears to be the causative factor.

In **paralysis of the phrenic nerves** (innervating diaphragmatic respiration) or **paralysis of the costal nerves** (innervating thoracic respiration) the expansion of the thorax is much limited, hence the pul-

monary branch of the vagus is stimulated and the group of motor nerves which are intact call their mechanism into increased play to compensate for the inactivity of the other group.

Modified Variations.—Many of the causes of increased respiratory frequency only manifest themselves when some extra demand is made upon the respiration, such as exertion, which in health would not embarrass the breathing at all. Especially is this the case in those conditions where it depends upon an insufficient oxidation and compensated heart lesions.

In **chronic lesions** which would otherwise tend to induce rapid breathing the process may be so gradual that the system adapts itself to the disability. In **tuberculosis**, for example, the lessened lung area is frequently offset by the reduction of body weight, thus lessening the necessity for oxygen and CO_2 elimination.

CO_2 Narcosis.—In all those cases where increased frequency is caused by accumulation of CO_2 in the blood, this condition if prolonged sufficiently to induce CO_2 narcosis will eventually slow the respiration.

Brain Compressions and Toxemias produce a diminuation in the frequency of respiration.

Variations in Rhythm are also quite common in health. It is difficult to assign any definite or single explanation for the physiologic irregularities that are frequently seen, sometimes even in sleep, especially among children and nervous people. It is highly probable that they depend largely upon central and reflex disturbances of the inhibitory and accelerator factors of the nervous mechanism, of the same kind, only differing in degree, as those inhibitions of the centre which arise from powerful emotions and cause prolonged variations in rapidity.

Cheyne-Stokes Breathing is the most notable example of pathological abnormality in respiratory rhythm. In this type of breathing there is a rapid cresendo and diminuendo, both as to frequency and depth, the paroxysms lasting from thirty seconds to a minute, and intervened by a pause in respiration. The cause of the phenomenon is unknown, but it doubtless has its explanation in some disturbance of the central nervous system. It is usually of grave import. In children, however, it occasionally occurs physiologically during sleep.

Cabot describes a very irregular gasping respiration, accompanied by a nodding of the head, "the chin being thrown quickly upward at each inspiration, and falling slowly during expiration," which he attributes to nervous dissociation, and regards as a precursor of death. This is seen in **Uremia**, and certain **Diseases of the Brain.**

Asthma is a common illustration of disturbed rhythm; the exciting cause is usually reflex, as described under **Reflex Inspiration.**

Variations in Amplitude or Depth are quite common within physiological limits, depending upon practically the same conditions that determine the rapidity of the process.

THE VASCULAR SYSTEM.

General.

The Circulation.—The blood of the body is contained in a closed set of tubular vessels, through which it is propelled by the beat of the heart; the coats of the vessels are muscular, elastic, and microscopically permeable. The blood leaves the ventricle of the heart by one artery of large lumen (aorta), which, in its passage to the peripheries, subdivides continually into more numerous arteries of less caliber but of a constantly increasing total capacity. At the peripheries the arteries quite suddenly subdivide into innumerable capillaries, of very small caliber, which with equal suddenness widen out again into the veins, and these, by a reverse progression to that of the arteries, viz., by coalescence into a less and less number of veins of increasing caliber, finally unite in two large veins (venae cavae) and empty again into the heart. The vessels lie in numerous planes, changing with the various movements and postures of the body, and their contents are of course subject to the influences of gravity. This combination of conditions gives rise to all the dynamic and static phenomena of the circulation.

If to the above set of premises we add that the blood carries oxygen and nourishment to the tissues, removes waste gases and products, and equalizes the heat of the body, and that the muscular features of the system are under the control of various nerve

centers and fibers, we have the data for the physiological rationale of the vascular function.

The Blood.—Histologically the blood consists of a fluid (plasma) in which are floating cellular bodies known as the corpuscles. These comprise—

(1) Red Corpuscles.
(2) White corpuscles.
(3) Blood plates.

Of these the white corpuscles are the only true cellular structures, with nuclei and stroma. The red corpuscles contain nuclei at the early stages of development of the fetus, but they disappear at the seventh month. In certain conditions of excessive formation of red corpuscles in the blood—e. g., after excessive hemorrhage—these nucleated red corpuscles may be seen in goodly numbers. The blood plates are homogeneous, elliptical bodies, whose only function, so far as known, is to assist in coagulating the blood.

Reaction and Specific Gravity.—The normal reaction of the blood is alkaline, due no doubt to the preponderance of carbonate of potassium and phosphates in the plasma. This alkaline reaction is a necessary factor in the maintenance of life. Specific gravity is about 1060, that of the corpuscles being greater than that of the plasma; the red corpuscles have the greatest specific gravity.

Red Corpuscles.—Chemically the properties of the blood resolves itself into a study of the properties of the red corpuscles, as they are the only specialized

constituent of the blood, whose chemistry plays an important part in the physiology of the circulation.

The red corpuscles are by far the most numerous of the microscopic elements of the blood. Separately their color is a pale yellow, but in large numbers they give the red hue to the blood. The proportion of the number of red to that of white corpuscles is as 1,000 to 1. The average number of red corpuscles in health is 4,500,000 to 5,000,000 to the square millimeter.

Function of Red Corpuscles.—The red corpuscles carry oxygen to the tissues from the lungs. This function they perform by virtue of a chemical constituent in their make-up, called hemoglobin, which forms a very ready but exceedingly loose combination with oxygen, thus forming an ideal carrier of this gas.

Hemoglobin.—This forms the most important constituent of the red corpuscle. Its exact chemical composition is unknown, for it is understood in that form it exists in the normal corpuscle. It is not in solution, nor is it in crystal form. Corpuscles in which the hemoglobin is intact are relatively opaque; blood in which the hemoglobin has been discharged from the corpuscles into the plasma (laked blood) although of a far darker crimson is relatively transparent. Hence it is probable that its normal condition in the corpuscle is one of solidity.

When hemoglobin combines with oxygen the resulting compound is known as oxyhemoglobin, and

is of a bright scarlet color, giving to arterial blood its characteristic vivid red. This compound readily parts with its oxygen to the tissues which lack it, and the remaining chemical is called reduced hemoglobin.

Hemoglobin combines with carbon dioxid independently of its bonds with oxygen, the former probably uniting with some other constituent of the hemoglobin than the latter. On the other hand, the taking up of carbon dioxid appears to increase the readiness with which the hemoglobin parts with its oxygen. These conditions give bases for two physiological processes: (1) The hemoglobin carries oxygen to and carbon dioxid from the tissues simultaneously; (2) the further along the process is continued, the more carbon dioxid the hemoglobin removes, the more readily it delivers oxygen. Both these statements of course are limited by the capacity of hemoglobin to take up both oxygen and carbon dioxid.

Anaemia.—The essential morbid element of anaemia is a diminution of the number of red corpuscles in the blood, associated with important abnormalties in the character of these corpuscles. Sometimes the number is scarcely lessened at all, but the amount of hemoglobin contained by them is greatly reduced. Nucleated red cells are usually formed in great quantities. Some of these are undeveloped forms of red blood corpuscles, doubtless due to nature's attempt

to repair the rapid destruction of red corpuscles by hurrying out new ones. Others (megoblasts) resemble blood plates but are considerably larger. Their significance is unknown.

Hemolysis.—It has already been stated that the form in which hemoglobin exists in the red corpuscles is unknown. It is known, however, that the hemoglobin is maintained in this combination by virtue of the osmotic pressure in the surrounding plasma, and a lowering of this osmotic pressure, by diluting the plasma, results in a discharge of the hemoglobin from the corpuscle into solution in the plasma. This discharge of the hemoglobin is called hemolysis.

Hemolysis may also be effected by the action of certain drugs and toxins upon the corpuscles themselves, destroying the stroma and liberating the contained hemoglobin. Notably is this result produced by the action of the serum of one animal upon the red corpuscles of an animal of a different species—the influence of the various species toward one another varying greatly in intensity. Moreover, this influence is artificially transferable; i. e., by repeatedly injecting into the blood of one animal the serum of a second whose hemolysis reaction is exceptionally strong toward a third, the blood of the injected animal will presently become as positive in its hemolytic reaction upon the third as is that of the animal whose serum was injected.

Transfusion.—The discovery of the hemolytic in-

fluence of one species of blood toward that of another species has resulted in an abandonment of the practice of blood transfusion from animals which for a time seemed likely to obtain as a means of repairing hemorrhages. The measure is still occasionally practiced from human being to human being, but this is almost entirely superseded by

Normal Saline Infusion, an infusion of salt and water, mixed in such proportions (one-tenth of one per cent of salt) as to exactly correspond to the osmotic density of the plasma.

Drugs and Toxins.—Certain drugs have marked hemolytic action on the blood, notably ammonia and chloroform. So also have numerous toxins, of which the venom of the snake is a conspicuous example. To this toxic hemolysis must be attributed most of the secondary anæmias resulting from infectious diseases and malignant growths.

* * *

Metabolism of Red Corpuscles.—There is a continual production and destruction of red corpuscles, the former process going on in the red marrow of the bones, where nucleated erythroblasts by Karyokinesis beget daughter cells containing hemoglobin. The nucleus is lost before the cells pass into the circulation. The scene of their destruction is a matter of question. For a long time it was thought that the spleen was the graveyard of red corpuscles, but later investigations make it probable that they are disintegrated and dissolved in the bloodstream. No hema-

tin is found in the splenic venous blood, as would be the case if hemoglobin were disintegrated there; and the occasional finding of a partly disintegrated red corpuscle in a leucocyte of the spleen is probably explicable by post-mortem ingestion, owing to the immobility of the red and the amoebic motility of the white corpuscle after death.

Repair of Hemorrhage.—After severe hemorrhages the watery part of the blood is quickly replaced by absorption from the tissues, and an excessive proliferation of red corpuscles takes place in the red marrow to make good the loss. These corpuscles are hurried out into the circulation without waiting for destruction of their nuclei; hence nucleated red corpuscles are found in the blood after severe hemorrhages.

Saline Solution.—One of the beneficial effects of an injection of normal salt solution in cases of hemorrhage and shock is probably that it keeps the remaining red corpuscles circulating more rapidly and promote their usefulness as oxygen carriers. (Howell.)

Anemia.—The same is true in any condition where red corpuscles are destroyed in large numbers, as in secondary and pernicious anemias, as long as the proliferating capacity of the marrow holds out. In these disorders the marrow is usually found in a dark red congested condition, due to excessive functionating.

Influence of Altitude on Red Corpuscles.—Removal of a mammal to a high altitude is always followed in

a comparatively short time by an increase in the count of red corpuscles, but whether this increase is real, or only proportional, i. e., whether there is an actual increased proliferation of red corpuscles or whether they become more concentrated owing to removal of water from the plasma, is a disputed point. In either case the phenomenon is doubtless a provision of nature to compensate for the rarity of the oxygen in the atmosphere, and we must suppose that this same rarity of oxygen operates as the necessary stimulus to set this compensatory process in motion.

The White Corpuscles.—The white corpuscles, as already stated, are the only true cellular type of body among the blood elements, possessing a nucleus, and all the other typical structure of a true cell. Their numerical proportion to the red corpuscles in normal blood is as 1 to 750-1,000, there being from 4,500 to 7,000 to the cubic millimetre. The number varies under different physiological conditions. They are most numerous during digestion, exercise, pregnancy and other conditions of increased physiological activity.

Varieties of White Corpuscles.—There are several different varieties of white corpuscles in normal blood, distinguished by their histological anatomy and also by their function. The latter distinction divides them into two classes:

(1) Lymphocytes.
(2) Leucocytes.

The characteristic differences between these two classes are that the former have no granules and very little ameboid faculty; the latter exhibit granules and their power of ameboid locomotion is so marked as to gain for them the nickname of the "wandering cells."

Subdivisions of these two classes indicate various stages of their development, lymphocytes being recognized as small and large lymphocytes, and leucocytes as transition (uninuclear), jolynuclear, and heart cells; the last named variety is exceedingly few.

Functions of Leucocytes.—The lymphocytes form but thirty to thirty-five per cent of the total white corpuscles, and their function is unknown. Many physiologists regard them as the immature forms which later develop into leucocytes.

The polynuclear leucocytes are the only class of whose function we have any understanding. As might be expected from their histology, their function is an elementary one as compared with that of the red corpuscles. They stand, as it were at the lower gateway of the circulation and (1) protect the tissues from foreign and pathogenic bacteria and (2) furnish proteids to the plasma. They also aid in the absorption of fat and peptones from the intestines, hence their proliferation during digestion, and assist in the coagulation of blood. The latter can hardly be classed as a physiological function.

Diapedesis.—Leucocytes possess the ability to penetrate the coats of the arteries and vessels. This faculty they exercise constantly in health and much more markedly in certain pathological conditions.

Coagulation of the Blood.—The actual process of coagulation of blood is familiar, at all events in its microscopic phases, to everyone. In a few minutes after it is shed, the fluid becomes first of a viscous consistency, then sets in a jelly-like mass, the clot shrinking during the process and pressing out a clear straw colored liquid called serum. This serum consists of the plasma, less a hypothetical substance called fibrinogen, which has gone to form the clot. Hence the metamorphosis may be expressed histolog- Dr Atkinson 20p July 21 Stolze Twelve—— ically as follows:

Plasma	Serum Fibrinogen Fibrin-ferment	Fibrin	Clot
Cells	Red Corpuscles White Corpuscles		

Process of Coagulation.—Considered more minutely the process consists of the rapid formation of a stringy substance called fibrin, in the form of a network of threadlike strands, which catches the corpuscles in its meshes and, shrinking, compresses them into a clot, squeezing out the serum. The red corpuscles, being immobile, remain in the position they are caught in; the motile leucocytes, on the contrary, may wriggle out of the meshes and be found in the serum. If the blood be stirred during coagulation

some red corpuscles may be broken away from the network and the serum contain some of these cells.

If freshly shed blood be whipped briskly with a rod the fibrin, as it forms, will cling to the rod, and may all be removed from the blood, which will then remain fluid and is called defibrinated blood.

Theories of Coagulation.—Many have been the experiments made for the purpose of determining the nature of the process, and many the theories built up on these experiments and the matter is still far from satisfactory elucidation.

It is generally conceded that coagulation depends upon the presence of an albuminoid substance in the plasma, to which the name of fibrinogen is given, for when blood is heated sufficiently to coagulate this element, the coagulating power of the blood is gone, and the power is restored by the addition of artificially prepared fibrinogen. This fibrinogen (a soluble substance) is supposed to be converted into fibrin (an insoluble compound) by the action of a ferment elaborated after the blood is shed, and called fibrin-ferment or thrombin. It has also been shown that soluble calcium salts are necessary to the process, for blood received into a solution of oxylates which precipitates these salts, does not clot.

It is assumed that the ferment necessary to the clotting process is derived from the disintegration of leucocytes and blood plates, and as these cannot be supposed to disintegrate to any extent until after the blood is shed, this theory accounts for the failure of

blood to clot within the vessels. It also probably explains why introduction of foreign bodies into the living vessels causes coagulation, and why shed blood coagulates faster when so disposed as to come in more extensive contrast with the sides of the container.

Anemia.—In pernicious and secondary anemias, where large quantities of red corpuscles are destroyed, hemoglobin is liberated. This hemoglobin destroys the white corpuscles, producing fibrin-ferment, which coagulates the blood and gives rise to **thrombi, emboli, petechial** and **capillary hemorrhages.**

Ligatures. Aneurisms.—The influence of a foreign substance in inducing coagulation is taken advantage of in surgery for the purpose of arresting hemorrhages and curing aneurisms by means of ligatures. The constricted circle acts as a foreign body and induces coagulation in the vessel, thus forming a natural plug. In order to be effective the ligature must rupture the inner coat of the vessel.

BLOOD PRESSURE.

Velocity and Pressure.—Two elements enter into the mechanical features of circulation, namely, velocity of the blood flow, and blood pressure. The former represents the amount of force that is expending itself in the direction of the axis of the vessel, driving the blood along it and may be measured at any point by inserting a bent graded tube parallel to the axis of the vessel and noting the height to which the blood rises. The latter represents the force expended in a direction at right angles to the axis of the vessel, and may be measured at any point by inserting a tube at right angles to the axis and noting the height to which the blood arises.

Factors.—The factors which take part in and modify these mechanical phenomena are (1) the heart beat, (2) the friction in the vessels, (3) the resistance of the vessel walls, (4) the constriction or dilatation of the vessels by vasomotor influences.

The heart beat, which throws the blood into the vessels and distends them, may be regarded as furnishing the total power or force. A part of the force thus exerted is employed in overcoming the friction inside the vessels; another portion is utilized in neutralizing the centrifugal resistance of the vessel wall, exerted in a radiating direction; (this centripetal resistance is due partly to the passive opposition of the walls themselves, and partly to their active mus-

cular constriction by vaso-motor influences); the remainder of the force is represented by the velocity of the blood stream.

Thus, all that part of the force supplied by the heart beat which goes to neutralize resistance to the blood stream represents blood-pressure; that which is in excess of resistance represents the velocity of the current.

Variations in Velocity.—From what has been said it is evident that the velocity of the blood current varies directly as the force of the heart beat and inversely as the resistance, and as the latter differs in different parts of the vascular channel the velocity naturally exhibits corresponding variations in these several locations.

As already stated, every successive subdivision of the primal artery (aorta) into arteries of smaller caliber involves an increase in the total circumferential area of the channel; in the capillaries the total area is estimated at 800 times that of the aorta. This means that the blood is subjected to a progressive increase of peripheral resistance and a corresponding diminution of its velocity. By the time it passes through the capillaries and enters the veins its velocity is very much reduced; thereafter, however, it undergoes a reverse process in regard to area, namely, a successive passage into a less and less number of vessels, of larger caliber, representing a progressively decreasing total area; and furthermore, it gets more and more into the suction influence of the intra-

thoracic negative atmospheric pressure. (See Chapter on Respiration.)

The relations thus described between the velocities of the various parts of the vascular channels, and all other relations, are subject to variation at any moment by the action of various conditions influencing the blood flow or peripheral resistance. Marcy sums the matter up in two general laws: (1) Velocity is influenced directly by the force of the heart and inversely by resistance. (2) Pressure is influenced directly by both the force of the heart and resistance; is indeed the resultant of the two.

The velocity in the arteries is temporarily increased and diminished during systole and diastole; the variations are called systolic and diastolic velocities, and the average between the two, the mean velocity. This mean velocity, as indicated, varies for each artery.

The velocity in any channel can of course be modified by the constriction or dilatation of the vessel by the vaso-motor mechanism, thus relieving or increasing peripheral resistance.

Variations in Pressure.—From the foregoing it is plain that blood pressure, like velocity, is different in different parts of the vascular system. Generally speaking it may be said that it varies in about a coincident manner with velocity, as far as the arteries and capillaries are concerned.

Thus while the increasing peripheral resistance due to the multiplication of vessels tends to increase

pressure, the ever diminishing heart impetus, due to its distribution over an increasing area and its expenditure in overcoming resistance, furnishes a more than corresponding reduction of the other factor in pressure, with the net result that the pressure in the capillaries is lower than in the arteries, although the resistance is greater.

In the veins, however, this coincidence between velocity and pressure is lost. From causes already explained, the velocity of the blood stream increases from the capillaries to the heart, and the pressure, on the contrary, steadily diminishes, owing to decreasing resistance (less and less vessels) until the pressure in the venae cavae is practically nil.

The pressure in the arteries, like the velocity, is increased and diminished during systole and diastole, the difference being the mean arterial pressure.

Pressure is also increased by constriction and diminished by dilatation of the vessels through the vaso-motor mechanism, the former increasing and the latter diminishing peripheral resistance.

Adjustment of Pressure.—The well-being of the body demands a more or less constancy of blood pressure, and, more urgently still, demands the maintenance of a minimum pressure. This condition is preserved by means of a physiological adjustment between heart beat and resistance, sometimes effected by the heart, sometimes by the vaso-motors, often by both.

Thus in digestion, when the abdominal vessels con-

tain an extra amount of blood, and during muscular exercise when the blood is largely determined to the muscle-vessels, the fall in pressure which would otherwise occur in the other vessels is forestalled by an increase in the heart's action. Other examples will occur to the student, or will appear in discussing other functions.

High Blood Pressure results from any pathological condition which (a) increases the volume of blood in the vessels; (b) increases the force of the heart beat; or (c) offers abnormal resistance to its passage. Conditions which increase the total volume of blood are rare, the increase usually occurring either on the venous or arterial side, at the expense of the other.

Simple Cardiac Hypertrophy throws an abnormally large quantity of blood into the aorta at each systole, at the expense of the veins, thus raising arterial and lowering venous pressure. The impetus with which the blood is propelled also contributes to the increase of pressure.

Drugs and Toxins of certain kinds exaggerate the force of the heart and thus raise pressure. Most of these agencies also cause constriction of the vessels, which contributes to the increase of pressure.

Nephritis raises blood pressure primarily by vasoconstriction in the kidney, thus increasing peripheral resistance, and secondarily by toxic sclerosis of the artery walls, abolishing their expansibility.

Arteriosclerosis increases the rigidity, hence the

resistance of the vessel walls, and thus raises pressure.

Low Pressure.—Conversely, any influence which (a) weakens the heart beat, (b) lessens the volume of the blood, or (c) diminishes resistance to its passage, lowers pressure.

Myocarditis, Dilatation and **Fatty Degeneration** of the heart are attended with low arterial pressure, both because of diminished impetus and because an abnormally small quantity of blood is thrown into the aorta at each systole.

Hemorrhage lowers pressure by taking blood out of circulation.

Low Fevers (typhoid, typhus, etc.) and **Constitutional Diseases** which inhibit nutrition without any sclerotic changes in the blood vessels, rob the vessel walls of their tone, thus abolishing their elastic recoil and lowering pressure.

PULSE.

The Pulse.—If the vascular system consisted of a set of rigid tubes, each discharge of blood from the ventricle would push the whole mass of blood forward and simultaneously empty from the venae cavae into the auricle a similar quantity of venous blood. The elasticity of the aorta wall makes it easier to distend the artery than to move the whole mass of blood forward, and this is what happens primarily to make room for the blood discharged by the heart beat.

As soon as the semi-lunar valves close, the elastic coat of the aorta recoils, and drives the columns of blood onward. This succession of distension and recoil passes down the entire arterial system, in the form of a wave, diminishing as the total area of the channel increases, until in the capillaries, where the total area suddenly increases many times, the wave is lost. This wave of distension and recoil constitutes the pulse and, for the reason described, is only seen in the arteries.

Abnormal Pulse.—The normal propagation of the pulse wave may be interfered with by (a) conditions within the heart, or (b) conditions in the vessels. Such interference may make itself manifest in (a) the amplitude, (b) the regularity, (c) the velocity of the wave. These factors constitute the character of the pulse. The pressure in the vessel is frequently

included in the character of the pulse, but is really an entirely different phenomenon to that of the pulse proper, pressure being a static condition at any given moment, while the pulse is a dynamic condition requiring time as one of its elements.

Hypertrophy.—In general, anything which increases the force of the heart beat increases both the amplitude and velocity of the pulse wave. Hypertrophy of the heart, especially of the ventricle, in its early effects, is one of those influences. Later, when compensation fails, the amplitude of the wave is diminished and generally the velocity also. But it must not be forgotten that the effect of increased heart force on the wave may be offset by conditions of the vessels.

Myocarditis and Fatty Degeneration weaken the muscular force of the heart, and therefore diminish both amplitude and velocity of the pulse-wave, unless offset by vascular conditions.

Mitral Insufficiency, as long as compensation is good, has no perceptible effect upon the pulse wave, as the disturbances are all back of the ventricle.

In **mitral stenosis** the amplitude and velocity of the pulse wave is diminished because of the less quantity of blood poured into the ventricle at each systole.

Aortic insufficiency has a very characteristic effect upon the pulse wave. The hypertrophied condition of the ventricle augments the force behind the pulse and increases its amplitude, giving a high incline of

ascent, but the decreased resistance offered by the leaky valve to the aortic recoil induces a rapid, almost sudden collapse of the artery, giving a steep incline of descent. This excessive backward collapse may even be seen in the capillaries, evidenced by capillary pallor at each diastole. This pulse is called the Corrigan pulse, and is pathognomonic of aortic insufficiency. It may, of course, be absent in this lesion, if conditions of the vessels offset the valvular influence. (Quincke pulse.)

In **aortic stenosis,** on the other hand, the amount of blood thrown into the artery is smaller than normal, in spite of the hypertrophy of the ventricle, hence the amplitude of the pulse wave is reduced. The smallness of the pulse in contrast with the force of the ventricle, is very characteristic of this lesion.

Failure of Compensation.—In all of the valvular lesions, as soon as compensation fails, all of the characteristic effects of the compensated lesions disappear and the effect common to them all becomes one of enfeebled heart force, namely, small amplitude and low velocity.

* * *

Velocity of Pulse Wave.—As already stated, in a rigid system of tubes the pulse wave inaugurated by the discharge from the ventricle would be instantaneously propagated to the venae cavae. Hence, it follows that the velocity of the pulse wave is in direct proportion to the rigidity of the artery wall. It is, of course, in inverse proportion to the volume

of blood concerned, and therefore, to the calibre of the vessel. The velocity of the pulse wave must be carefully distinguished from the velocity of the blood stream, and from the rapidity of the heart beat.

Catacrotic Pulse Waves.—The elasticity (elastic recoil) of the arterial walls produces some modifications of the arterial pulse wave just described, by means of reflected waves set in motion by the recoiling wall of the artery. These are called catacrotic waves. The most significant of them is the dicrotic wave, which occurs immediately after the recoil has started, and noticeably interrupts the downward incline of the pulse. It is caused as follows: When the aorta begins to recoil it propagates the blood in both directions, toward the peripheries and toward the heart. The latter organ has shut its semilunar valves against which the proximalward wave strikes and is reflected as a distalward wave, once more producing a slight arterial distension.

Exaggeration and Diminution.—In extremely rigid conditions of the artery walls, as in **arteriosclerosis** and **atheroma**, the effect of the secondary wave is, of course, hardly perceptible, and in these conditions, therefore, there is a notable absence of the dicrotic wave. The same is true when the blood in the artery is under high pressure, so that the artery wall is already, by virtue of the fundamental wave, under tension, hence in cases of high arterial tension the dicrotic pulse is diminished.

On the other hand an easily distensible artery and

a low blood pressure favors the perceptible effect of the secondary wave, and in these conditions the dicrotic wave is very noticeable—**e. g., typhoid fever, tuberculosis, malignancy,** etc.

In **aortic insufficiency,** of course, the dicrotic pulse is absent, because at the recoil of the artery the blood, instead of striking the semi-lunar valves and producing a secondary wave, passes through the leaky valves into the ventricle. The collapse of the pulse, as already stated, is sudden, with no catacrotic waves.

THE HEART-BEAT.

Theories of the Heart-Beat.—Two opposing theories hold the field in regard to the cause of the heart-beat. One, the neurogenic theory, holds that the contraction of the heart muscle is due to the influence of nerve cells contained in the muscle; the other, the myogenic theory, attributes the function to an inherent property of rhythmic contractity possessed by the muscle itself. The strongest argument in favor of the neurogenic theory seems to be the presence of nerve cells in the heart tissues, as no experiment or observation has ever demonstrated that these nerve cells take any part in the origination of the heart-beat. For the myogenic theory it may be said that it can be demonstrated that the wave of contraction follows the musculature, and also that it can be made to travel in either direction at will—a fact which is incompatible with nervous control in the light of the polarity of neurons. The only serious objection to the myogenic theory, namely, the absence of muscular connection between the auricle and ventricle, has recently been removed by the demonstration of the auriculo-ventricular bundle, or bridge of Hiss.

Automaticity of Heart-Beat.—Whatever may be the difference of views as to the origination of the heart-beat, there is unanimity of agreement that it is an automatic function, i. e., that it is independent of the cerebro-spinal nervous system, and that al-

though the central nervous system exercises a certain species of control and regulation of the heart's action, it has nothing whatever to do with the causation of its rhythmical beat. For the heart continues to beat when it has been completely cut off from all connection with the nerve centres; and contractions which have ceased in a heart thus isolated may be reinaugurated.

The Heart-Beat.—The muscular contraction which results in the heart-beat begins in the muscular coats at the mouths of the venae cavae and the pulmonary veins, spreading over the auricles it causes a simultaneous contraction of the two auricles, thence passes by the bundle of His to the ventricles, which also contract simultaneously. This is the end of the heart-beat, and a pause intervenes between its conclusion and the commencement of the next contraction wave, called the period of rest. The period during which the auricles are in contraction is called the auricular systole, the time they are relaxed the auricular diastole. Corresponding periods for the ventricles are called ventricular systole and diastole. The passage of the contraction wave over the heart is accompanied by the passage of a simultaneous electric wave, the potential of the muscle in contraction being higher than that of the muscle in relaxation.

Heart Block.—In rare instances a pathological condition is seen consisting in an interruption, by means of a tumor, gumma, degeneration, or other de-

structive process, in the muscular isthmus between the auricle and ventricle (the bundle of His). The functional aberration resulting from this lesion is a disconnection of the auricle and ventricle in regard to their rhythmicity, each maintaining a rhythmic beat of its own, and is known as heart block. Sometimes the block is complete, in which case the rhythmicity of the auricle and ventricle bear absolutely no relation to each other; in others the block is only partial, and the respective rhythmicities then bear some regular proportion to each other, e. g., the ventricles will beat twice to the auricle's once.

HEART ACTION.

Change in Form and Position of Heart During Beat.—The contraction of the heart muscle of course diminishes, and probably in health completely obliterates the cavity of the ventricle, driving the contained blood into the artery. The change in the form of the ventricle thus produced differs with the position of the heart, i. e., with the position of the body, but in general it is decreased in its vertical and transverse, and increased in its dorso-ventral diameters. The spiral arrangement of the superficial muscles causes a rotation of the ventricles in systole. This rotation compensates for such vertical shortening as occurs, and maintains the apex of the heart in its normal position against the chest wall, while the sudden contracting of the heart muscle produces the apex impulse. The impulse is also augmented by the sudden straightening of the curved aorta by its distension with blood.

Hypertrophy.—When from any cause, such as increased resistance or forced acceleration, the heart muscle is hypertrophied, one of the earliest and most characteristic manifestations of its hyperthophy is an exaggeration of the apex beat against the chest wall, especially in cases (most common) where the hypertrophy either begins in or is confined to the ventricles.

This phenomenon is seen in those valvular diseases

which produce ventricular hypertrophy, viz., insufficiency and stenosis of the semilunar valves, as well as in so-called idiopathic hypertrophy.

Heart Sounds.—The heart beat is accompanied by two sounds, corresponding to the ventricular systole and diastole, succeeded by a pause corresponding to the period of rest. The first sound is a dull booming character, the second of a sharper tone, and a minor third higher in pitch. The following occurrences are synchronous with the first sound and probably enter into its composition: (1) Closure of the auriculo-ventricular valves. (2) Contraction of the heart muscle. That the first of these elements is not the sole factor is proved by the occurrence of the first sound in a bloodless beating heart, and by the booming nature of the sound. The second sound is doubtless caused by the closure of the semilunar valves, as it disappears on hooking back these valves in the living heart.

Careful experiment has shown that the first sound occurs at the beginning of the systole, and the second at the end of the systole (diastolic).

Hypertrophy.—In hypertrophy of the heart muscle in the early stage, the hypertrophy is accompanied by an exaggeration of its contractility, manifested by an exaggeration of the muscular element of the first sound. Later, when compensation begins to fail, this gives place to an enfeeblement of the first sound, which, however, retains a more boom-

ing character than normal because of the greater volume of muscle involved.

Mitral Insufficiency.—The mitral valves close at the beginning of the ventricular contraction, to prevent any of the blood contained in the ventricle being driven back into the ventricle. If the mitral valves are "insufficient," i. e., if they leak, at each ventricular systole some of the blood is driven back into the auricle (regurgitation). This backward current, meeting the oncoming stream in the auricle, causes a whirl of blood, which, impinging on the edges of the valves, produces a peculiar blowing sound, called a **murmur.** This murmur, of course, occurs at systole, and is a **systolic murmur.**

If, on the other hand, the mitral orifice, from any cause, such as **thrombosis** or **inflammatory vegetation,** is stenosed, the passage of the blood from auricle to ventricle is impeded, and the rush of liquid under increased tension through the narrow orifice produces a rippling sound, never so loud or blowing as in mitral leakage. This sound, of course, occurs just prior to ventricular systole, and is called a **presystolic murmur.**

Aortic Insufficiency.—When there is a leakage of the semilunar valves of the aorta, at each recoil of the aorta the blood is partially driven back into the ventricle and, meeting the oncoming stream from the auricle, produces a sound similar to that produced in mitral leakage, but it is, of course, heard following systole; in other words, during diastole,

and is called a **diastolic murmur.** The first heart sound is indistinct (though loud) because of the overdistension and slow contraction of the ventricle.

Aortic Stenosis.—In stenosis of the aortic orifice the blood is forced from the ventricle under increased pressure through a very narrow opening, and a sound is produced similar to that described in mitral stenosis. It occurs, of course, during ventricular contraction, and is a systolic murmur.

The sounds produced by the lesions above mentioned are propagated along the direction of the blood current and as the valves themselves all lie within a very small area the sounds are best differentiated by listening to them at the surburban heart points, to which they are transmitted. Thus the sounds and their significance may be classed as follows:

(1) Systolic murmur, heard best at apex, indicates mitral insufficiency.

(2) Pre-systolic murmur, heard best at apex, indicates mitral stenosis.

(3) Diastolic murmur, heard best at aortic arch, indicates aortic insufficiency.

(4) Pre-diastolic murmur, heard best at aortic arch, indicates aortic stenosis.

Pulmonary Lesions.—The valves of the right heart are subject to precisely the same conditions as here described, and present a corresponding set of sounds, but their occurrence is so rare as to demand no special attention.

Haemic Murmurs.—Similar dynamic conditions to those of valvular insufficiency may be brought about by abnormalities of the blood, which decrease its density, and murmurs are frequently thus produced. Particularly is this the case in anemia. The sounds are neither so loud nor so constant as in valvular lesions, and are called haemic, or functional murmurs.

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Accentuation.—Anything which augments the force of the heart beat increases the muscular impetus and therefore accentuates the first sound of the heart. We have already seen that **hypertrophy** is the chief of these influences; **exercise, emotions,** and **certain drugs** also have this effect, and it is a not infrequent practice among diagnosticians to administer strychnia in order to augment the first sound and bring out suspected abnormalities.

The second sound, depending upon the closure of the semilunar valves, is accentuated by anything which increases the intra-arterial pressure, and its accentuation is pathognomonic of diseases in which peripheral resistance is increased, of which **arteriosclerosis** and **kidney diseases** are familiar illustrations. Accentuation of second sound is, of course physiologic in **old age,** because of atheroma of the vessels.

Accentuation may occur from the causes described in any of the valves separately, which together make up the respective sounds. These separate accentua-

tions must be diagnosed by auscultating at the outlying points of sound transmission for the separate valves.

Reduplication of the heart sound is due to the asynchronous occurrence of the events which produce it. In the first sound, of course, the valvular element is the only one which can be reduplicated and as this element is completely overshadowed by the muscular element, its reduplication is practically unrecognizable.

Reduplication of the second sound is not an infrequent symptom and is usually due to some **pathological condition of the coronary arteries.** The two sides of the heart, being unequally nourished, do not functionate synchronously; the semilunar valves close asynchronously, and give a double sound.

The same phenomenon results from an unequal tension in the two ventricular cavities, due to **valvular leaks** and **stenoses.**

Myocarditis and Fatty Degeneration reduce the force of the muscle contraction and therefore make the first sound weak and indistinct. The second sound, by contrast, seems accentuated; but in cases of high blood pressure from other causes, the second sound may of course be genuinely reduplicated.

The Cardiac Cycle.—At the instant that the wave of muscle contraction begins, at the mouth of the pulmonary veins, the left auricle is full of oxygenated blood which has been poured into it by the pul-

monary veins. As the contraction passes over the auricle its capacity lessens, the pulmonary valve is closed by the pressure of the blood, and the latter is driven forward through the auricula-ventricular valve into the ventricle. The contraction wave now passing over to the ventricle, its capacity is reduced, the pressure closing the mitral valve and driving the blood out into the aorta. Meantime the auricle has relaxed and refilled with blood from the pulmonary veins. The blood being forced into the aorta distends its walls, which, however, promptly recoils and closes the semilunar valves. This ends the cycle. A precisely similar cycle of events takes place in the right auricle and ventricle, except that the blood received from the venae cavae is venous blood, and is pumped by the ventricle into the pulmonary artery.

The regular sequence of this cycle may be interfered with in many ways, or rather by many pathological conditions, since the nature of the interference is practically the same in every case, but owing to the compensating faculty of the heart, it is only after long and persistent interference that any actual change occurs in the cycles.

Mitral Insufficiency.—Thus, if the mitral valve leaks, at each ventricular contraction a portion of its contained blood is forced back into the auricle and an insufficient quantity pumped into the aorta. The auricle is then receiving blood both from the venae cavae and from the ventricle and becomes much distended, and the tendency is for a backward

stasis of circulation. However, the auricular muscle responds to this demand by hypertrophying (compensatory hypertrophy) and contracting more forcibly. In time, of course, hypertrophy can no longer make up for the increased work, and compensation fails. The result, as one would suppose, is backward stasis in the veins, high venous and low arterial pressure, the former causing dropsy and CO_2 poisoning, the latter insufficient oxygenation, shortness of breath and general atony. (See Respiration and Elimination.)

Aortic Insufficiency.—Leakage of the semilunar valve produces, by the same dynamic process, a compensatory hypertrophy of the ventricle and eventually backward stasis.

Mitral Stenosis.—Stenosis of the mitral orifice, although a reverse condition to insufficiency, produces the same results by a somewhat different mechanism. Here the narrowness of the opening imposes a systolic pressure upon the auricle, which is compensated by auricular hypertrophy, later producing backward stasis.

Aortic Stenosis.—Narrowing of the aortic opening operates upon the ventricle precisely as vertical stenosis does upon the auricle, with the same backward train of events.

Leakage and Stenosis of the Right Heart.—Insufficiency and stenosis of the valvular mechanism of the right heart produces a set of conditions precisely corresponding to those described for the left heart.

In these cases, however, the pulmonary circulation is the first to feel the effects of backward stasis due to failing compensation, and respiratory difficulties are the earliest and most direct results. Fortunately, as already stated, they are much rarer, owing to the less opportunity for functional derangement than in the systemic circulation.

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The Coronary Arteries.—Another source of interference with the cardiac cycle is frequently seen in a lesion of the coronary arteries. In order to properly carry out their function all the heart muscles must themselves be regularly and adequately supplied with nutrient blood, and any condition of the coronary arteries (e. g. sclerosis, embolism, thrombosis) will produce a disturbance in the performance of the heart cycle. Sudden stoppage of the heart (in diastole, of course) often results from this cause, which is also thought to be the explanation of the phenomenon known as angina pectoris.

Intrapulmonary Pressure.—Any condition producing an increase of intrapulmonary pressure will, if continued long enough, embarrass the right ventricle, and bring about its hypertrophy, with eventual failure of compensation and fatal stasis. **Emphysema** is a notable example of this. (See Respiration.)

Systemic Pressure, long continued, such as is caused by **chronic Bright's disease, diabetes, arterio-**

sclerosis, etc., will bring about the same train of results in the left heart.

Tonicity and Maximal Contractions of Heart Muscle.—The heart muscle, like the skeletal muscles, is in a constant state of more or less contraction; unlike the skeletal muscles, however, it appears to possess this tonicity independently of its connection with any nerve centre. It exhibits a further dissimilarity to skeletal muscles in that its contractions are always maximal, i. e., when it contracts at all it contracts to the farthest limit of its contractility.

NEUROLOGY OF HEART.

Nervous Control of the Heart.—Although the origination of the heart's action is independent of the central nervous systems, its performance is largely modified by two sets of efferent neurons. One, the vagus, is received from the undulla, and inhibits the action of the heart muscle, slowing its beat, and cancelling its tonicity, so that when the heart (as occasionally happens) is stopped by stimulation of the vagus, it is arrested in exaggerated diastole. The other set, the accelerators, come from the sympathetic chain, and augment the velocity of the beat.

Cardio-Inhibitory Function.—Stimulation of the vagus nerve inhibits the heart's beat and cancels the tonicity of its muscle, finally arresting it in exaggerated diastole. The activity of this nerve also lessens the conductivity of the heart muscle, causing a condition known as heart block, i. e., where the contraction wave does not regularly pass over from the auricle to the ventricle, so that there are two beats of the former to one of the latter. Experiment proves that the fibres of the vagus reach and influence both the auricles and ventricles direct, but their distribution to and influence upon the ventricles are less than to the auricles, while with the accelerator nerves the reverse is the case.

Heart Block.—In this condition, already described, the beat of the ventricles is always in excess of that of the auricles, because of the distribution of accel-

erator and inhibitory influence above referred to, and also because of interference with the conductivity of the heart muscle described above.

Reflex Inhibition.—The cardio-inhibitory action of the vagus may be called into play reflexly by stimulation of various sensory neurons, notably by the stimulation of those sensory fibres of the vagus which are distributed to the thoracic and abdominal viscera.

The cardio-inhibitory centre is in a constant state of tonicity, acting as a continual automatic drag on the heart, preventing it from beating as rapidly as it would otherwise do, and this tonicity of the centre is doubtless a reflex phenomenon, mediated apparently by various sensory impulses.

Accelerators Balance.—The accelerator nerves, derived from the sympathetic, whose influence is precisely the opposite to that of the vagus, are also capable of being called into action by reflex means. Thus it seems that the velocity of the heart is normally regulated by the influence of two antagonistic nerve-currents, one accelerating and the other inhibiting its beat. It appears probable, however, that the inhibitory mechanism is most often invoked, and that even acceleration is more frequently the result of inhibition of the function of the vagus than of stimulation of the accelerators.

DIGESTION.

Mastication is performed by means of the digastric muscle, which depresses the jaw; the masseters, temporals, and internal pterygoids, which raise it; and the external pterygoids, which move the jaw laterally (grinding).

Innervation.—All the muscles of mastication receive their motor power by way of the inferior maxillary branch of the fifth cranial nerve.

Bulbar Paralysis.—One of the earliest manifestations of bulbar paralysis is **dysarthria**, or difficulty of jaw movement, due to involvement of the root of the fifth nerve. The lesion of this particular part is probably no earlier in fact than that of other bulbar areas, but its impairment is noticed first. Later, there is complete inability to masticate.

Imperfect Mastication, from whatever cause, sends the food to the stomach imperfectly prepared, thus delaying its passage through that organ and giving rise to fermentation, flatulence, and indigestion, and is, in these days of hurry, a fertile cause of stomach trouble.

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Salivary Glands.—These consist of the parotid, submaxillary and sublingual glands, all of which belong to the type of tubular glands.

The Saliva is a colorless viscid liquid of alkaline reaction and a specific gravity of about 1.003. Its principal ingredient is an enzyme called ptyalin,

which reacts upon starch to produce a diastase. It also contains some proteid maltose, and sodium potassium and calcium salts. In solution saliva contains carbon dioxid, the product (and measure) of the metabolic activity of the glands.

The secretion contributed by the parotid gland is richer in ptyalin and poorer in mucin than the secretion of the other two; the latter is given a more alkaline reaction than the parotid secretion.

Ptyalism is an excessive secretion of saliva. It is rarely, if ever, a primary complaint, but depends upon some other pathological condition, and the saliva is usually altered in character as well as increased in quantity.

Inflammations of the Mouth and Throat, unless accompanied by a high temperature, are always attended by an increased flow of saliva, due partly to vaso-dilator conditions and partly to increased reflex stimulation. In such cases it is usually acid in reaction, because of the increased absorption of CO_2 and other metabolic acid products.

Pregnancy is attended by a more or less degree of ptyalism.

Mercurial Ptyalism is due to hyperstimulation of all the salivary glands by the drug.

In Fevers the watery part of the saliva is rapidly absorbed by the mucous membrance to compensate for the general anhydrous condition of the tissues and the secretion is therefore thick and viscid, and feels dry and sticky. The same condition is found,

and for the same reason, in **Diabetes** and certain forms of **Nephritis.**

The Reaction becomes acid in **Fevers, Diabetes, Gout, Rheumatism and Nephritis,** because of the absorption of metabolic acid products, chiefly CO_2 in the first two diseases and uric acid in the others. In gouty subjects the acidity is sometimes so high as to erode the chin and corners of the mouth.

Innervation.—The salivary glands receive their stimuli both from the cerebral centres through the chorda tympani and from the sympathetic by way of the cervical ganglia. Experiment shows that cerebral stimulation produces a thin, watery secretion, poor in solids, whereas sympathetic irritation causes the secretion of a viscid thick substance, rich in solids.

The present theory is that the cerebral fibers mediate the purely secretory element in the function, i. e., the osmotic filtration of the gland, while the sympathetic performs a trophic part, increasing the metabolism of the cells and producing organic products.

Vaso-Motor Influences.—In addition to the above nervous supply, the chorda tympani carries vaso-dilator fibers, whose stimulation dilates the capillaries of the glands, and the sympathetic carries vaso-constrictor fibers whose stimulation constricts the capillaries.

Dry Mouth is a condition, described first by Hutch-

inson, in which the secretion of the saliva is inhibited as the result of a central nervous disturbance. The parotid glands become hard but painless.

In fevers the watery portion of the saliva is rapidly absorbed by the mucous tissues to compensate for the general anhydrous condition of the body, and the saliva is therefore thick and viscid.

* * *

Reflex Mechanism.—The function of the salivary glands is a reflex one, whose centre is in the medulla. It receives afferent stimuli from numerous sources, chief among which, of course, are the sensory of the tongue and palate, by means of the glossopharyngeal and lingual nerves. The stomach by means of the vagus, and the nose by means of the olfactory nerve, also furnish afferent stimuli, and that the cerebral centres may both directly stimulate and inhibit the salivary centre is evidenced by the well known effects of various emotions and ideas upon the secretion. (Watering at the mouth and parched throat, due to longing and fear respectively.)

Experiment demonstrates that the efferent part of this reflex is mediated wholly by the chorda tympani, the sympathetic of itself being ineffectual to do so.

Stomatitis and Glossitis.—Undoubtedly the ptyalisms of inflammations of mouth and tongue are partly due to irritation of the afferent fibers of the glosso-labio-laryngeal nerves.

Gastric Ptyalism, as seen in catarrhal gastritis, gastric ulcer, before vomiting, etc., are due to stimu-

lation of the afferent fibers in the gastric branch of the vagus.

Deficient Saliva is one of the results of imperfect and hasty mastication, because the latter act is one of the chief exciters of the salivary reflex.

The Function of the Saliva is both mechanical and chemical. Mechanically, it moistens the food, enabling it to be conveniently masticated and swallowed, and dissolves certain parts of the material so as to act upon the taste buds.

Chemically its principal function is to convert the starch in the food into dextrin and sugar by means of its diastase, ptyalin. This is supposed to be accomplished by the starch molecules taking up water and splitting into more elementary molecules. The reaction is not completed in the mouth, but the food, thoroughly mixed with saliva, passes into the stomach and remains there some little time untouched by the gastric juices, while the diastase finishes its work.

Heat increases the activity of the salivary process up to about 40 degs. C. At 65 degs. C., however, the ptyalin is destroyed.

Cold decreases its activity; at 0 deg. C. it is inhibited.

Acids destroy the activity of ptyalin, even so small a percentage as .003 free hydrochloric acid being fatal to it.

Boiled starch is much more amenable to the diastase than raw starch, as the starch takes up some

water in boiling, and the shells of cellulose surrounding its grains are broken up.

Imperfect Salivation.—When mastication is hurried and food is bolted, the diastatic action of the saliva is not well begun before the stomach is reached. This delays the stay of the food in the stomach, causing fermentation and flatulency.

When food is coated with an impermeable layer of fat or other substance upon which saliva has no action, the same ill result occurs.

Diluted Saliva.—When drinking is indulged in at the same time as eating, the saliva is diluted and the intensity of its diastatic action greatly weakened. This is a frequent cause of indigestion.

Inhibition.—The behavior of ptyalin under extremes of heat and cold is sufficient indication of the evils of eating food too hot or too cold, which contains starch or of drinking extremely hot or cold fluids during eating.

Altered Reaction.—Inasmuch as the action of ptyalin is favored by an alkaline medium and inhibited by acids, it is easy to understand the poor diastatic quality of the saliva in fevers, diabetes, rheumatism, gout, nephritis, in which the reaction of the secretion is acid from containing CO_2 and uric acid.

Deglutition is partly a voluntary and partly an involuntary reflex act. It is usually divided into three

stages, which, however, depend upon anatomical rather than physiological differentiation.

First Stage.—This embraces the passage of the substance swallowed from the mouth to the pharynx, and is a voluntary motor phenomenon. It is performed by the levator muscles of the tongue, which elevate that organ against the soft palate and drive the morsel between the anterior pillars of the fauces. At this point voluntary control of the act ceases, and it enters the

Second Stage, including the passage of the morsel through the pharynx. The pharynx being primarily a respiratory passage, and containing the entrance to the larynx (glottis) it is essential that the passage of food be as rapid as possible through this region. The contraction of the mylohyoid muscle in the floor of the mouth drives the morsel through the pharynx into the esophagus, the elevation of the tongue against the hard palate preventing any retrograde movement. This action is assisted by the hyoglossal, and by the contraction of the constrictor pharyngeal muscles. Other muscles are called into play to prevent the sidetracking of food into the air passages. The levator and tenson palati shut off the nasal cavities; the laryngeal opening is closed by the adduction of the vocal cords by the cricoarytenoids and constrictor glottis, and by the elevation of the larynx. Experiment demonstrates that the epiglottis does not lie down flat during deglutition, as formerly believed, but remains erect.

Third Stage.—The passage through the esophagus into the stomach varies according to the physical condition of the food. Liquids fall directly through the esophagus by gravity, and there await the arrival of the peristaltic muscular wave incited by their presence, which opens the sphincter of the stomach. Solids are moved downward by this peristaltic contraction and pass directly into the stomach. The wave of constriction is furnished by the successive contraction of the circular muscles, the longitudinal muscles, contracting immediately in advance of the circulars, dilate the tube in readiness for the oncoming bolus. The sphincter of the stomach, which is normally in a state of closure, opens upon the arrival of the muscular wave, and admits the food into the stomach.

Dysphagia (difficulty in swallowing) of course results from any impairment of any of the above described factors in deglutition, or from any condition making the performance of their part in the act painful.

Inflammation of any portion of the alimentary tract from the mouth to the stomach, including **glossitis, pharyngitis, tonsillitis,** and **esophagitis,** make swallowing difficult because of the pain caused by the contact of the food and by muscular contractions.

Ulcers, Growths, etc., have the same effect for the same reason.

Spasms of the passages offer an obstacle to swallowing, both by closing the passage and by tempo-

rarily paralyzing the musculature. In spasms, occurring during the act of deglutition the food is immediately regurgitated.

Innervation of Deglutition.—First Stage.—The inauguration of the act is mediated by the twelfth cranial nerve, the motor nerve of the tongue, which controls the process as far as its entrance upon the second stage, is the passage of the morsel into the pharynx. Thereafter the act is a true reflex.

Second Stage.—The afferent stimulus of the pharyngeal division of swallowing is mediated by the sensory fibers furnished to the mucous membrane of the pharynx by the glossopharyngeal and trigeminus. The efferent impulse is carried by the motor fibers of the same nerves.

Third Stage.—The esophageal stage of the act is stimulated afferently by the sensory fibers of the vagus and superior laryngeal, and efferently by the motor fibers of these nerves and by the special accessory.

The Centre, as already stated, resides in the medulla, near the nuclei of the neurons concerned.

Bulbar Paralysis results in voluntary dysphagia due to involvement of the medullary root of the twelfth cranial nerve, one of the most distressing symptoms of this lesion. The same lesion of course paralyzes the reflex mechanism of the second and third stages of deglutition, but these are subordinate to the first or voluntary stage.

Choking.—In bulbar paralyses and central lesions involving sensory paralysis of the laryngeal nerves, the stimulus which protects the laryngeal opening at the glottis is wanting, and particles of food frequently enter the larynx and cause partial or complete asphyxia.

Movements of the Stomach.—The fundus or cardiac end of the stomach is not concerned in the muscular movements of the organ. It is simply, so far as its musculature is concerned, a continuation of the longitudinal fibers of the esophagus, expanding for the better reception of the food. It must be remembered that normally the cavity of the stomach is precisely in accordance with its contents, being in a state of collapse or distension as it is empty or full of food.

The pyloric end of the organ is the persistaltic portion. Here, a few minutes after the entrance of food, waves of contraction in the transverse and oblique fibers are set up, running distalward, and pressing the food against the pyloric sphincter as soon as any of the contents of the pylorus are ready, physically and chemically, the proper reflex stimulus is furnished, which opens the sphincter and ejects the prepared food into the intestine. Cannon believes that this stimulus depends upon the consistency of the food, since solid substances passed against the pylorus prevent relaxation, while liquid food taken into the stomach passes very quickly

into the intestine. The point is, however, far from settled.

Fermentation and Flatulence.—When the motor mechanism of the stomach is so interfered with as to delay the passage of food through it, the carbohydrates of the food are fermented by the action of the bacteria of fermentation, forming organic acids (lactic, butyric, acetic). These in turn form acid gases, that are eructated through the mouth. The occurrence of these fermentations is shown by the establishment of the **hyperacidity of the stomach** due to excess of the acids mentioned.

The conditions which most markedly diminish the motility of the stomach and thus produce fermentation are **dilatation, pyloric obstructions, malignant growths,** and **chronic gastritis.**

Excessive Food may also interfere with the muscular movements of the stomach, and produce the above train of symptoms, except that in such case the acidity of the stomach contents will be relatively below par, as enough is not secreted to take care of the excessive amount of food.

Digestibility of food is usually gauged, so far as the stomach is concerned, by the length of time it remains in the stomach, and as it is discharged into the duodenum as soon as it is liquified, this is equivalent to estimating it by the ease with which the stomach reduces the food in question to a fluid.

Atony of Stomach Walls.—Due to distension, (1) putting greater strain on muscles, (2) compromising

the vessels and lowering nutrition, and (3) dragging organ downwards and kinking pylorus.

Pyloric Stenosis, producing first hypertrophy and late dilatation of muscular walls.

Defect in Nutrition of stomach walls.

Disease of Stomach Walls.—Gastritis, Carcinoma, etc.

Passive Congestion of Stomach, interfering with nutrition.

"Splashing" Sounds.—As already stated, the normal stomach is always distended in exact proportion to the amount of solid or liquid food it contains, its muscular coat contracting tightly around these contents and promptly expelling all air and gas that may be present or formed. If the muscular walls be atonic, however, the stomach will sag under the weight of liquid; gas and air will be present, and upon shaking the patient the contents may be heard to "splash." This is pathognomonic of gastric atony. Its exhibition in patients apparently free from stomach trouble is explained by the fact that in early stages of atony the stomach is still able to empty itself regularly, and therefore gives no signs of gastric indigestion.

Pyloric Insufficiency is seen sometimes in muscular diseases and malignant infiltrations, the contractile power of the pylorus being lost and the food pouring through the orifice too quickly. Its results are, of course, intestinal indigestion; one of the most noticeable symptoms being a regurgitation of gas produced

by the action of the alkaline juices of the duodenum on the acid chyme.

Muscular Innervation.—The stomach is furnished with fibers from the vagus nerve and the splanchnic ganglia of the sympathetics, but as its muscular contractions continue after section of both these groups of fibers it is hard to escape the conclusion, that the stomach muscle is automatic either by virtue of an intrinsic property of the muscle itself or of the nerve ganglia which richly invest it.

However the movements of the stomach may originate, they are controlled and regulated by the vagus and splanchnic, both of which furnish sensory and motor fibers to the organ. Experiment shows that the efferent effects of the vagus are directly motor, while those of the splanchnic are inhibitory, causing dilatation and relaxation.

Through these two media the movements and secretions of the stomach can be modified or inhibited, either reflexly or by the influence of the higher centres.

Eructation is a motor reflex, mediated as to its afferent stimulus either by the sensory fibers of the gastric vagus in the stomach itself, or by the glossopharyngeal nerves.

Vomiting is a reflex, having its centre in the medulla (q. v.). Its afferent paths are numerous. Brunton enumerates them as follows: Pharyngeal branches of glosso-pharyngeal; pulmonary and gas-

tric branches of vagus; gastric branches of splanchnic; renal, mesenteric, uterine, ovarian and vescical nerves. Its efferent paths are the motor fibers of the gastric vagus and splanchnic.

The centre is amenable to influences direct from the brain (**central vomiting**); and to those of certain conditions of the blood, as in **infectious diseases.**

Paresis of the Stomach Walls is a very rare occurrence, but is occasionally met with. The result is an acute dilatation of the stomach, due to the complete abolition of the "tonal" energy.

Spasm of the Cardia and Pylorus is a reflex phenomenon, due to irritation of the stomach nerves by hyperacid contents. It produces what is known as **pneumathosis,** the gas in the stomach being caught between the two spasmodic contractions and distending the stomach, causing great pain.

Dyspnea and Cardiac Failure.—Any sudden or serious distension of the stomach may produce dyspnea by undue pressure on the diaphragm and faintness and palpitation by interference with the dynamics and innervation of the heart.

Any condition which irritates the gastric nerves can disturb the heart and respiration reflexly through the vagus nerve. This is especially common in **hyperacidity** of the stomach.

Stomach Juice.—The gastric secretion is a thin colorless liquid, specific gravity about 1.002, acid reaction and of a characteristic odor. Its essential in-

gredients are hydrochloric acid, pepsin, and rennin, but it also contains mucin, inorganic salts, a trace of proteids, and some lactic acid.

The percentages of its ingredients vary, of course, under different physiological conditions. The average percentage of HCl. in a normal stomach is 0.3. It is supposed that the acid is derived from the sodium chlorids in the blood, by double reactions, liberating free HCl. At the beginning of stomach digestion the acidity of the juice is somewhat neutralized by the alkaline saliva with which the food is mixed. The acidity of the juice as secreted, however, is constant, in health.

Anacidity.—In acute and chronic gastritis in which there is excessive mucous secretion, the activity of the cover cells is interfered with and a deficiency in hydrochloric acid results. The cause here is local. Later there is atrophic or cirrhotic destruction of the gland cells, resulting in complete failure of HCl. and pepsin.

In Anemia, Infectious and Wasting Diseases there is a deficient secretion of HCl. due to constitutional causes, probably nervous in character.

In Carcinoma of the stomach there is usually a complete absence of free HCl., due to both local and constitutional interference with cell activity.

Excessive Eating may produce temporary anacidity of the gastric contents, on account of the inability of the stomach to secrete a relatively sufficient amount of acid for the quantity of food.

Hyperacidity may be due to increase of HCl. or of organic acids, as lactic acid.

In **gastric ulcer** the secretion of HCl. is occasionally increased by the hypersensitiveness and irritation of the cells surrounding the ulcer. More often the irritative effect of the hyperacidity causes the ulcer.

In the early stages of **gastritis** (dyspepsia) there is hypersecretion of HCl. from the same causes.

Excess of lactic, butyric and acetic acids result from fermentation of carbohydrates. These fermentations occur where there is delay in the stomach due to another impairment. Hence they are found in **dilatation, organic obstructions of the pylorus, malignant infiltrations,** etc.

Where lactic acid is constantly excessive and HCl. constantly absent, **carcinoma** may be usually diagnosed.

Neurotic hypersecretion occurs in neurosis of the stomach, such as neurasthenia, hysteria, and may be, constant or periodic. The increased acidity frequently irritates the sensitive sensory nerves of the stomach, causing sensations of hunger and through them the solar plexus, causing gastralgia, both of which are usually relieved by food.

* * *

Innervation.—The nervous control of gastric secretion is mediated from a medullary centre by means of the vagus nerve. It is undoubtedly as a rule a reflex act, whose afferent stimuli are derived

from various sources. Animal experiments demonstrate that the smell, taste, and even the sight of food operate as afferent stimuli to the secretory centre, depending of course on the conscious state for their effectual influence. Pawlow calls the secretion thus induced a "psychical secretion," and it is certain that this type of stimulus plays an important role in the ordinary process of digestion.

It is also probable that afferent stimulus is furnished by the sensory nerves of the stomach itself, set up by the presence of food, and that this type of stimulus is independent of consciousness, being mediated by the sensory fibers of the vagus.

Experiment shows, however, that the character of this stimulus is not mechanical, but due to the chemical properties in the food. Different foods possess the stimulating power in differing degrees, and some lack it altogether, and can only be digested if the psychical stimulus be present to start secretion. Thereafter these foods, in their altered, partially-digested condition, are capable of setting up the stimulus.

Afferently the secretory centre is amenable to both stimulus and inhibition from the higher centres direct, as the emotions are potent factors in modifying gastric digestion.

Nervous Dyspepsia is regarded by Leube as a group of cerebral impressions made by the irritative effect of an ordinary digestive process upon hypersensitive nerves of the stomach, and the eructations

which occur in this condition as a form of motor reflex from such irritation. Another afferent phenomenon is the hypersecretion of HCl. referred to under the preceding section, causing sensations of hunger and gastric pain, relieved by food.

1. **Glands Containing Chief of Peptic Cells.**—These are located mainly at the pyloric end of the stomach and secrete pepsin and rennin, the digestive enzymes.

2. **Glands Containing Cover Cells.**—These are massed in the middle or pre-pyloric part of the stomach and furnish hydrochloric acid. Peptic cells are found intermingled with them.

In Chronic Intestinal Gastritis the chief and cover cells are both largely replaced and supplanted by a proliferation of mucoid cells, the result being a diminution of normal secretion and an outpouring of mucous, hence indigestion. Later there is either atrophic or sclerotic degeneration, resulting in complete absence of gland cells.

In Chronic Interstitial Gastritis, the outpouring of mucous gives the same sensation of hunger, but the appetite is very quickly appeased on account of the hypo-secretion of gastric juice.

Indigestion (i. e., an abnormal length of time necessary for stomach reduction of food) may result from eating unpalatable or insipid food, owing to the deficiency of "psychic" secretion in the stomach.

Pepsin-Hydrochloric-Acid Digestion.—Pepsin is an enzyme which, like other enzymes, is modified in its action by temperature, high temperature up to 40 degs. C. hastening, and low temperature retarding its activity. Unlike the enzyme, ptyalin, however, its activity requires the presence of an acid; hence pepsin digestion in the stomach is the joint result of pepsin and hydrochloric acid.

Pepsin converts proteids by a series of metabolic steps into peptones. Howell indicates this process by the following landmarks of transformation: Proteid, acid albumine, primary proteoses, secondary proteoses and peptones.

The process is not always completed, and the chyme may be ejected into the duodenum in any one of the above stages of transformation, whence it appears that pepsin digestion is not so important in itself as in its preparatory influence on the food looking to further digestion by the pancreatic juices.

Fat is not, as a rule, influenced by pepsin and hydrochloric acid, except that the breaking up of the proteids frees the fats from their combination with the proteids and prepares them for intestinal digestion.

Rennin Digestion.—So far as is known, the only action of this enzyme is to convert the casein of milk and other casein-containing foods into paracasein, by a process commonly known as curdling, which we must suppose is beneficial to the ultimate

digestion of the casein, although the rationale of the process is at present obscure.

Gastric Absorption.—Experiment demonstrates that very few foodstuffs undergo absorption in the stomach. Water, formerly thought to be readily absorbed, is known to pass through almost unchanged. Salt solutions are unabsorbed in any less concentrated solutions than 3 per cent. Sugars and peptones are absorbed, but with difficulty.

Acidosis in Diabetes.—Bainbridge, in The Lancet, points out that the onset of coma is usually preceded for a day or two and accompanied by a fall in the output of the acetone bodies, the excretion of b-oxybutyric acid may sink from 15 grammes or more to as little as one gramme, and the acetone and diacetic acid may be either absent or present only in traces. The decreased excretion of acetone in the breath also indicates that the production of acetone bodies is actually lessened and that the kidneys are not able to carry out their functions. The fall in the output of sugar and nitrogen, the fall of body temperature, and the lessened intake of oxygen all point to a decline in the total metabolism. Equally characteristic features of coma are the diminished alkalinity of the blood and its diminished content of carbonic acid.

Acid intoxication, in the strict sense of the term—that is, poisoning by hydrogen ions—never occurs, since the tissues never lose their alkaline reaction; the term is justifiable, however, if it is used to de-

scribe the metabolic disturbances resulting from the abnormal production of acids in the body or from the administration of mineral acids accompanied by a fall in the alkalinity of the blood and tissues. The tissues are very sensitive to even a slight diminution in their alkalinity, and the acute intoxication produced by mineral acids is believed to be due to a fall in the alkalinity of the blood and tissues or, in other words, to a diminution in the concentration of hydroxyl ions relative to the hydrogen ions; this has been proved to occur as far as the blood is concerned.

* * *

Intestinal Movements.—The intestines are supplied with both circular and longitudinal muscles. The former furnish what is known as peristalsis, by means of the successive contraction of the circular muscles distalward, the effect of which is to drive the contents of the bowel onward. Some physiologists hold that this wave of peristaltic contraction is preceded by a wave of inhibition. That the progression of the wave depends upon the arrangement of the musculature is shown by the fact that resection of a piece of the intestine and replacement in the opposite direction is followed by reversed peristalsis in the portion whose polarity is thus changed.

Diarrhea and Constipation represent the two opposite abnormalities of intestinal peristalsis, the former being due to increased and the latter to diminished peristalsis.

In Intestinal Obstruction, if complete, the peristalsis above the seat of obstruction is increased by the accumulation of feces, and eventually the pressure overcomes the distalward movement and fecal vomiting ensues.

Peristaltic Innervation.—Experiment and observation make it probable that the muscular activity of the intestines, like that of the stomach, is automatic in origin, and regulated by the cerebro-spinal and sympathetic nerves. In both of these relationships it is undoubtedly a reflex act, in the former case mediated by intrinsic ganglia, in the latter case by a spinal centre. The stimulus is doubtless a mechanical one, normally furnished by the entrance into the bowel of the food matter.

The Small Intestine is innervated, both afferently and efferently, by fibers of the vagi, and sympathetic fibers from the dorsal vertibrae via the splanchnic and semilunar ganglia.

The Large Intestine is innervated in its upper portions by the same nerve supply as the small intestine; in its lower sections (descending colon and rectum), it receives fibers from the second to the fourth sacral nerves, and from the inferior mesenteric ganglia of the sympathetic.

All of the cerebro spinal centres concerned in the intestinal movements are subject to direct stimulation and inhibition by the higher cerebral centres. The effects of emotions and ideas upon bowel centres. are familiar phenomena.

Diarrhea and Constipation, representing respectively increase and diminution of intestinal peristalsis, are almost invariably due to a disturbance of the nervous reflex governing this muscular function.

Diarrhea, the expression of an exaggerated peristalsis, is most frequently due to a **catarrh**, either primary or secondary, which renders the intestinal nerve ends irritable and leads to overstimulation of the peristaltic reflex.

Psychical Diarrhea, such as accompanies sudden emotions, is doubtless brought about through vasomotor mechanism. Certain emotions, such as fright, inhibit the splanchnic vaso-constriction, causing a congestion of the splanchnic vessels and overstimulation of the intestinal reflexes.

Intestinal Dyspepsia is attended with diarrhea, because the undigested food acts as a foreign irritant to the intestinal nerve ends and excites peristalsis. Undigested food is found in the stools. (Lienteria.)

In Typhoid Fever and Cholera the bacteria and their toxins focus as a rule in the bowels, producing intestinal catarrh and exciting peristaltic reflex. **Intestinal tuberculosis** operates the same way.

Gall Stones induce diarrhea by mechanical irritation of the intestinal nerves.

Worms excite peristalsis both by mechanical and chemical stimulus.

In Intestinal Obstruction the accumulation of material above the obstruction acts as an abnormally powerful mechanical stimulus and increases peris-

talsis, but naturally the increased peristalsis does not result in diarrhea. The bowel being greatly distended, the vigorous peristalsis can frequently be seen through the abdominal wall, and in connection with absence of defecation makes a characteristic syndrome, as well as a measure of the extent, of intestinal obstruction.

Exposure to Wet and Cold may produce diarrhea by means of vaso-motor reflex, the skin stimulus causing congestion of the splanchnic vessels and thus overstimulating peristalsis. Burns of the skin frequently act in the same way.

Gastric Indigestion causes diarrhea by reason of the insufficient liquification of the chyme, which therefore exerts too great a mechanical stimulus on the nerves of the intestine.

Reflex Nervous Diarrhea may result from reflex irritation from some other diseased organ.

Constipation, the expression of diminished peristalsis, is frequently due to faulty innervation of the peristaltic reflex, either in its afferent or efferent phase.

Neurasthenia, Hysteria, Anemia, Chlorosis, etc., are attended by constipation because of the diminution in nerve energy, coupled with a debility of the muscles of the intestines.

In Chronic Constipation the nerves lose their peripheral sensibility on account of neglect to respond to the reflex desire for defecation. Later the intestinal muscles become flaccid.

In certain spinal diseases such as **myelitis** and **meningitis,** constipation results from involvement of the reflex arcs.

All fevers in which there is no specific intestinal catarrh (as in typhoid, vida supra) are attended by constipation. This is due partly to the rapid absorption of moisture from the bowel contents, making their passage difficult, partly in diminution in the amount of contents, lessening the reflex stimulus, and partly to malnutrition of the intestinal muscles, or weakening their intrinsic power.

Diabetes is attended by constipation, due partly to rapid absorption of moisture and partly to diminution of bowel contents, much of their normal solid constituents being eliminated by the kidneys.

Constitutional Diseases (Tuberculosis, Syphilis, etc.) unless specifically attacking the bowel with catarrh, exhibit constipation, due to malnutrition of muscle and faulty innervation.

Intestinal Absorption.—The intestines are the chief absorbent portion of the alimentary tract. Their absorptive capacity may in general be said to range in precise ratio to the proximal topography—being greatest in the duodenum and diminishing until the least degree is reached in the rectum. This, however, is necessarily subject to modification according to the substances considered for absorption.

In the Small Intestine absorption is very active, and proceeds simultaneously with digestion. The

process is accomplished by two forces (a) diffusion and osmosis, and (b) the selective and absorptive energy of the epithelial cells in the intestinal wall.

In the Large Intestine the unabsorbed remnant from the small bowel remains a long time, during which digestion proceeds by means of the digestive juices derived from the upper bowel. No digestive juices are secreted in the large intestine, but absorption takes place, especially of water and proteids, the former accounting for the eventual consolidation of the feces.

The large bowel has an alkaline reaction, which favors the growth of putrefactive bacteria, so that putrefaction is a usual phenomenon in this locality.

The Pancreas, a glandular body of the tubular type (frequently called the salivary gland of the abdomen) secretes a thin watery fluid, alkaline in reaction, of a specific gravity about 1.007, of which the essential digestive ingredients are enzymes called Trypsin, pancreatic diastase, and lipase. The first breaks up proteids or rather the proteid products already prepared by the pepsin of the stomach; the second breaks up starch; the third fat.

Functional Pathology of the Pancreas is obscure and indefinite, owing to the fact, first, that its physiology is poorly understood and indissolubly blended with that of other organs; second, that its complete disablement is extremely rare and as long as partial integrity remains it performs its work with reasonable satisfaction.

Innervation.—The pancreas derives its nervous motive energy from the cerebrospinal and sympathetic systems by means of the vagus and fibers from the celiac plexus. Vaso-motor fibers play an important role in the proper functionating of the organ.

The stimulus is supplied in a rather unusual fashion. The effect of the acid contents of the stomach, when ejected into the duodenum, is the production of a substance called secretion, which, being absorbed by the blood and carried to the pancreas, stimulates it to secretion.

The functional pathology of the organ is further complicated and obscured by this nervous mechanism.

Trypsin Digestion.—Trypsin is not elaborated as such by the pancreas, but as a stable zymogen known as trypsinogen, which is converted into trypsin by an enzyme of the duodenum called by Pawlow enterokinase.

As already stated trypsin completes the work already begun by pepsin on the proteids. While pepsin works only in the presence of an acid, trypsin works best in an alkaline medium. The action of trypsin is much more rapid and thorough than that of pepsin. The proteids, which are assumed to contain two groups of molecules, the hemis and the antis, are split up into primary and secondary proteoses, and thence into peptones corresponding to the proteid molecules hemi and anti-peptones. Trypsin, however, does not stop there, but breaks these

peptones up still further into tyrosin, leucin, aspartic acid, tryptophan, lysatinin, and other end products whose number and character of course vary with the length of time the food has been subject to tryptic digestion.

No definite nutritive significance has yet been assigned to these various products. Lysatinin is probably the source of some of the urea formed in the body.

Pancreatic Diastase Digestion is practically the same as that of ptyalin, namely, a hydrolytic action upon starch, converting it into maltose and dextrin. It evidently deals with such starchy elements in the food as escape the influence of the saliva.

Lipase Digestion comprises three stages: (1) The enzyme splits up the fats of the food, by a hydrolytic process, into glycerine and fatty acids. (2) The fatty acids combine with the alkaline salts present to form soaps. (3) The soaps are broken up into minute globules which do not coalesce (emulsification). The latter process is not due to the enzyme lipase, but to the physical properties of the pancreatic juice. Lipase is found in other tissues of the body where fats are concerned, notably the mammary glands, muscles, liver, etc. Its action is greatly assisted by bile.

Fat in the feces, to any abnormal amount, is usually regarded as presumptive evidence of pancreatic disability, but can only bear this diagnostic interpretation in the absence of symptoms pointing to sup-

pression of bile, intestinal tuberculosis, diarrhea, or large ingestions of fat. (See **Composition of Feces.**)

Lipuria (fat in the urine) is subject to the same provisional interpretation.

THE LIVER.

The function of the liver is threefold: (1) The secretion of bile, (2) the elaboration of urea, and (3) the formation and storage of glycogen.

Suppression of Bile is a frequent symptom in parenchymatous diseases of the liver, notably in **acute yellow atrophy, hepatic cirrhosis, malignancy, syphilis,** and **abscess.** It is manifested as a rule by a more or less severe **jaundice.**

Uremia, now generally recognized as a suppression of urea rather than its retention, is undoubtedly the direct result of abnormal hepatic metabolism although the exciting cause is the failure of the kidney to excrete urea. This phase of the hepatic function is too obscure to furnish any definite contribution to functional diagnosis.

Disordered Glycogen Metabolism, and consequent starvation of the tissues, especially the muscular tissues, accompanies almost every **degenerative disease of the liver.**

Coma due to retention and toxemia of metabolic products also accompanies all severe parenchymatous diseases of the liver, such as **yellow atrophy, cirrhosis,** etc.

* * *

Bile.—This important secretion performs a twofold office; one of an excretory and the other of a digestive character. Besides water and organic solids, it contains bile-pigments, bile acids, cholesterin,

lecithin, fats, soaps, inorganic salts, and a species of ancho-albumin erroneously called mucin.

Suppression or Retention of Bile is always accompanied by disturbances of intestinal digestion, especially of fat-splitting ferment whose activity it increases three-fold, and of the proteolytic and amytolytic ferments whose activity it doubles. These digestive disturbances are therefore always seen in conditions which interfere with the elaboration or discharge of bile, as **yellow atrophy, malignancy, hepatic abscess, cirrhosis, cholecystitis, gall stones,** etc.

Nervous Disturbances also attend bile suppression or retention, due to the suspension of the excretory office of the bile and the consequent re-absorption of pigments, cholesterin, licithin, and similar excretory materials.

Bile Pigments.—The bile contains two principal pigments, bilvinbin and bilverdin, the latter being an oxidation of the former. These pigments are derived from the hemoglobin of the blood as a product of the disintegration of red corpuscles, the iron which is separated from them being retained for the formation of new hemoglobin. The pigments are therefore in the nature of an excretion, and are passed largely with the feces in the form of urobilin and stercobilin. Some portion of them, however, is known to be absorbed in the intestines and re-secreted by the liver, though to what purpose is not clear.

Suppression or Retention of bile invariably manifests itself by an absence of these pigments from the feces; resulting in clay colored stools, common to all those diseases already enumerated as interfering with flow of bile.

Excessive Bile, on the other hand, exhibits an excess of pigments in the feces, producing dark green or very yellow stools, seen principally in **excessive proteid feeding** and in **gall stones** following on obstruction of the duct and consequent accumulation of bile.

* * *

Bile Acids.—These acids are organic and are two in number, glychocholic and torocholic. They do not occur as free acids but in combination with sodium basis as acid salts. They are formed directly in the liver cells, and are the elements in the bile which assist the pancreatic secretion in the digestion of fats. They are largely absorbed in the intestines and resecreted by the liver—probably because of their value as a stimulus to bile secretion.

Biliousness, as commonly understood by the laity, is due to the poverty of the bile in these acid salts. In their comparative absence fats are very hard to digest or to dispose of, hence the intolerance of the patient to greasy foods; and the stimulation of bile-flow is weakened, causing a general diminution of bile and bile-function.

Cholesterin and Lecithin are both of them true excretory products, being secreted from the blood

by the liver cells. They both pass out unchanged in the feces.

Innervation.—The secretion of bile does not appear to be under the control of any special set of nerves, but is a constant function, dependent for its regulation only upon the vaso-motor influences of the hepatic vessels, and probably also on the character of the blood flowing through them.

Discharge of Bile.—Normally the bile is not continuously given by the liver directly to the duodenum, but is stored in the gall-bladder, guarded by a sphincter, and discharged at intervals during digestion. The ejection of the chyme into the duodenum acts as a stimulus for the reflex which relaxes the sphincter, contracts the gall bladder, and throws the bile into the duodenum. This reflex is mediated by the vagus and splanchnic nerves. Bruns asserts that the stimulus depends on the character of the chyme; acids, alkalies, and starches being inert, proteids and fats or their products effective.

Retention of Bile results from any condition which (a) interferes with the proper performance of the gall-bladder reflex, or (b) obstructs the passage of the duct, in which case the bile pigments are absorbed and appear in the epithelial tissues as jaundice.

Familiar examples of the first variety are **malaria, sepsis, icterus neonatorum, pernicious anemia.**

Of the second class conspicuous instances are **gall-**

stones, cholecystitis, malignant growth, and **cirrhosis.**

Function of Bile.—Bile performs a two-fold role, (1) that of an excretory function, eliminating cholesterin, lecithin, and bile pigments, and (2) that of a digestive process, accelerating the emulsification and hydrolysis of fats in the intestine. The antiseptic properties formerly ascribed to the bile have been greatly discounted by recent observations.

Glycogen is frequently called animal starch, as it has the same general chemical formula as vegetable starch, and is amenable to the action of ptyalin with practically the same end-products. It is constantly present in the normal liver, being stored up during digestion and given out to the tissues gradually in the fasting intervals. The quantity present varies, therefore, in regard to the occurrence of meals; the mean quantity is about 2.5 per cent of the weight of the liver. It is held by the liver cells in a loose chemical combination much the same way as the hemoglobin is held by the red corpuscles of the blood.

Formation of Glycogen.—Normally the great bulk of glycogen is undoubtedly derived from the hydro carbon of the diet. These reach the liver in the form of dextrose, levulose, and saccharose, which are dehydrated by the liver cells into glycogen, as follows:

$$C_6H_{12}O_6—H_2O=C_6H_{10}O_5.$$

Experiment and observation make it clear that

glycogen may also be derived from the proteids of the blood. Of the various end-products of pepsin and trypsin digestion, those containing a nitrogen atom are probably converted in the liver into urea, the balance going to make sugar, which is subsequently converted into glycogen.

Fats increase the amount of glycogen in the liver, but whether by direct conversion into this product or by diminishing the consumption of glycogen by the tissues is not certainly known. Von Noorden asserts that glycogen is derived directly from fats.

An important factor in Diabetes is a deficient formation of glycogen. The sugars which are normally utilized for this purpose remain unchanged in the blood and are secreted through the kidney. Recent experience shows that this deficiency is a disability of the pancreas digestion rather than of the liver, although certain forms of liver disease are attended with glycemia.

Uses of Glycogen.—The modern doctrine of glycogen is that it represents the storage of the carbohydrate nutriment of the body which the liver holds in trust and gives out to the blood as required. Reaching the liver in the form of glucose these hydrocarbons are dehydrated, as explained, into glycogen and stored in the liver cells. When needed by the system they are inverted into glucose and given to the blood. It is now known that in order that the muscles and other tissues may take up and utilize the glucose distributed by the liver there

must be added to it a product of the pancreas. Just what this product is is not definitely known. Von Noorden assumes it to be a polymerising agent which enables the protoplasm to again invert the glucose into glycogen.

Diabetes.—It seems pretty well established that the prime factor in the glycocemia and glycosuria of diabetes is the failure of this polymerization process, so that the tissues, although flooded with sugar, are quite unable to assimilate it; hence it accumulates in the blood and is forced through the kidneys. This agrees with the two chief somatic conditions found in diabetes, namely, poverty of the organs and tissues in glycogen, and an excess of sugar in the blood; also with the clinical fact that increased muscular exercise does not reduce the glycemia, and points to the pancreas as the chief organ of offending in diabetes.

Diabetes Masked by Obesity.—In cases where this polymeric disability exists but there is no impairment of fat synthesis, the excess of sugar is utilized in making fat instead of passing into the urine. In these cases there is obesity and no glycosuria; it is a case of diabetes masked by obesity.

Regulation of Glycogen Formation.—The formation of glycogen is a constant metabolic process, and modified only by the quantity and quality of the diet. Each hydrocarbon has a different limit to

which the liver is able to dispose of its sugar-product. For starch, which forms so large a portion of our diet, no limit is known, and of the sugars themselves glucose has the highest limit, as much as 150 gms. being taken care of at one dose. When these articles are fed beyond their limit glycemia occurs, due to the surplus sugar-products which the liver is unable to store as glycogen.

Regulation of Glycogen Distribution.—That the doling out of glycogen in the form of glucose by the liver to the tissues is in some way under the control of the nervous system seems probable from the fact that certain disturbances of the nervous equilibrium bring about a temporary glycemia and glycuria, and also from Claude Bernard's famous "piqure" experiment, in which he induced glycosuria by a puncture of the fourth ventricle. The mechanism of this control is obscure. It is apparently mediated by a centre which receives its stimulus from the condition of the tissues (especially the muscles) in regard to sugar supply. An increased demand for sugar, as in increased metabolism (muscular exercise) brings about a reflex acceleration of its distribution from the liver, and vice versa. It would appear, however, that the inhibition reflex is only effective to a certain limit, as after periods of muscular rest, glycogen is found stored in the muscles, demonstrating that the supply is not absolutely regulated by the immediate demand.

In diabetic patients there is no doubt that this

stimulus continues to act upon the nervous center, since the tissues, although bathed in sugar are unable to utilize it, and are therefore hungry for it. Thus a vicious circle is effected of an organism already overburdened with useless sugar constantly stimulating the distribution of more.

Neurogenous Glycosuria is a temporary excess of sugar in the urine due to nervous shocks, neurasthenia, post-operative conditions, and other disturbances of nervous functions, and must not be confounded with true diabetes. There is no pancreatic disability in these neurogenic forms; they depend entirely on an exaggerated distribution of sugar by the liver—hence increased muscular metabolism will reduce the glycosuria.

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The Spleen is an organ whose function or functions are very obscure. It is supposed to (1) generate new corpuscles, (2) furnish a graveyard for red blood corpuscles, and (3) assist in the formation of uric acid.

Although a swollen spleen is the accompaniment of many diseases, particularly **typhoid fever, malaria, leukemia,** and **secondary anemia,** there is not sufficient data as to its physiological function to establish any diagnostic relationship between it and the disease in question.

ELIMINATION.

The Kidneys secrete urine. Inasmuch, however, as the constituents of the urine are all existent in the blood when it reaches the kidney, and are simply separated by those organs, it would be more correct to say that they excrete urine.

In the absence of one kidney, the other, if healthy, will compensate by performing double duty indefinitely. Complete suppression of renal function, however, rapidly produces death.

Process of Excretion.—Two principal theories hold the field as to the mechanism by which the kidneys excrete the urine:

1. **The Ludwig or mechanical theory** accounts for the process by the purely mechanical agencies of filtration and diffusion. In the glomeruli the entire urine, in a dilute condition, is filtered from the blood, the process being regulated by the pressure in the blood vessels. In the tubules it loses some of its water by diffusion and becomes more concentrated.

2. **The Bowman-Haidenhain theory** holds that the glomeruli separate water and inorganic salts from the blood, and the tubules secrete the organic constituents, urea, etc., both processes depending primarily upon the vital activity of the epithelial cells, although blood pressure necessarily plays a quite important part.

The preponderance of evidence is in favor of the latter theory, and it will be assumed here.

Albuminuria.—In their normal condition the epithelial cells of the kidneys are only permeable to those inorganic and organic constituents of the blood which make up the normal constituency of urine. In diseased conditions of the kidney which disable these cells, however, other elements are let through the glomeruli and tubules, chief among which is albumin. This phenomenon is known as albuminuria, and is common to all those diseases which injure the epithelial tissue of the kidneys.

There are two varieties of albumin found in pathological urine, Nucleo-Albumin and Serum Albumin. Of these the former is derived from the disintegration of the epithelial cells themselves, and is therefore not diagnostic of renal disease, as the disintegrated cells may come from other parts of the urinary tract. Serum albumen, on the other hand, is derived directly from the blood, and is evidence of the disability and permeation of the renal tissues.

The gravest forms of albuminuria, of course, are found in diseases of the kidney proper, i. e., in all forms of **nephritis, amyloid disease, tuberculosis, cancer, abscess, calculus,** etc.

The next severest occurrences of it are seen in those circulatory diseases, and diseases affecting circulation, which produce a secondary congestive effect upon the renal tissues, as **heart diseases, hepatic cirrhosis, tumors, anemia,** etc.

All **infections** and **toxic** conditions cause a transitory albuminuria by the extension of their poisonous action to the renal cells.

Hematuria (blood in the urine)—Is the result of grosser lesions of the kidney tissue, allowing blood en masse to enter the glomeruli and tubules. (This, of course, assuming that the blood originates in the kidney, and not in some other part of the urinary tract).

All of the causes above credited with the power of causing albuminuria may also, by affecting grosser injury, give rise to hematuria.

Hemoglobinuria is, of course, always present when hematuria is, but may occur independently in diseases in which there is great destruction of red corpuscles, e. g., **anemia, grave infectious diseases,** etc.

Glycosuria.—Ordinarily, that is to say under the pressure in which it normally exists in the blood, the renal tissues are impermeable to sugar. But when it reaches an abnormally high percentage it is excreted in the urine. This occurs in **diabtes mellitus** and is pathognomonic of that disease.

Casts are nothing more or less than particles of renal tissue which have acquired the mould of the tubules in passing through them, and in many cases gained a covering of epithelium. They are of numerous variety, depending upon their composition, or more often upon their appearance, such as hyaline, granular, waxy, fatty, and blood casts. True epithelial casts, composed entirely of epithelial cells,

are rare. Casts, of course, are found only in destructive processes in the renal tissues, and are diagnostic, when found in numbers, of organic kidney lesions.

Absolute anuria is rare, but may be caused by (a) complete destruction or disability of renal tissue, as in violent **acute nephritis** and in the last stages of organic kidney diseases, or (b) by complete pelvic obstruction, as in **nephrosis, calculus**, etc.

Oliguria (diminished secretion) results from any conditions injuring or disabling the secreting area and uncompensated by increased renal blood flow, as in **malignant diseases, amyloid degeneration, acute nephritis**, later stages of **chronic nephritis** after compensation has failed.

Polyuria (increased secretion) comes from conditions which while not completely disabling the secreting tissues, render them abnormally permeable to fluids, as in **interstitial nephritis, cirrhosis, diabetes**, etc.

* * *

Innervation.—The secretion of urine is a continuous process, performed, as stated, by the epithelial cells of the glomeruli and tubules, and is innervated by the sympathetic fibres supplying the kidneys from the renal plexus. The kidneys receive no nerves from the cerebro-spinal system, and their function is outside the province of voluntary acceleration or inhibition.

In view of the fact, elsewhere stated, that the

renal function is essential to life, it is fortunate that interruption of the motive power of urinary secretion, whatever that motive power may be, is so rare as to be practically unknown.

Increased and decreased secretion, however, may and frequently does result from nervous and emotional conditions, which must be explained by their effect upon the renal function through the nerve channels above described. It is worthy of note in this connection that such influences seem to affect only the gomerular portion of the function, since nervous and psychic polyuria consists always in an increase in the water and inorganic salts, with a corresponding drop in density, while the same types of oliguria exhibit simply a diminution in these ingredients with a corresponding rise in density. The organic secretions do not seem to be influenced in such purely nervous cases.

Hysteria, migraine, neurasthenia, epilepsy, delirium tremens are examples of nervous polyuria.

Post-operative shock, melancholia, cerebral disorders, etc., frequently inhibit the renal function, and may even cause death thereby.

Vaso-Motor Regulation.—It has already been said that even under the Bowman-Haidenhain theory the secretion of urine is partially regulated by the blood-pressure in the kidneys. This is brought about by vaso-motor mechanism. The kidneys are supplied

very richly with vaso-constrictor fibres, stimulation of which contrasts the renal vessels, raises pressure, lessens the flow of blood through the organs, and diminishes the secretion of urine. Inhibition of these constrictors dilates the renal vessels, increases the flow of blood, and accelerates secretion of urine.

Constriction of the arterioles at the skin, reflexly dilates those of the kidneys, increasing renal elimination.

Blood Flow.—From the foregoing it will be seen that the amount of secretion depends upon the quantity of blood passing through the kidney in a given time rather than upon blood pressure, and upon the latter only as it determines the former. High arterial pressure and low venous pressure, with no constriction of the renal vessels, will of course, drive large quantities of blood through the kidney and increase the urine. Low arterial pressure, even though the renal vessels be dilated, will lessen the flow of blood and diminish the urine.

Polyuria.—For the above reasons the quantity of urine secreted is always increased in those conditions which increase the flow of blood through the kidneys, as in **cardiac hypertrophy** primary (i. e., not due to contracted kidney) which induces high pressure in the renal arteries, and in **cystic degeneration of the kidney** and **hydronephrosis** which dilate the renal vessels.

Oliguria, or diminished flow of urine, is for like

reasons induced by all diseases which lessen the blood flow through the kidneys, e. g., **myocarditis, valvular diseases** (uncompensated), **lung diseases, hepatic cirrhosis,** which reduce the blood force, and in all diseases of the kidney itself which constrict the renal vessels.

The urine is normally a pale amber-colored fluid, slightly acid in reaction when passed, and of about 1.020 specific gravity. Its acidity depends chiefly upon the presence of acid phosphates, of which sodium phosphate is the most important, which, again, owe their preponderance to the proteids in the diet. The specific gravity is of course largely determined by the proportion of solids in solution, of which urea is the chief.

The average composition of normal urine (which of course varies under differing diets and physiological conditions) is as follows:

Water (average daily amount)	1500 c.c.
Urea (approximately)	30 grams.
Uric acid	1 gram.
Creatin	1.5 grams.
Sulphuric acid	4 grams.
Phosphoric acid	3 grams.
Inorganic salts	10 grams.
Pigments	Variable.

The specific gravity of the urine is, of course, **increased** by any influence which (a) increases the

amount of solids, or (b) decreases the quantity of water in the secretion.

Glycosuria (sugar in the urine) is by far the most important of the first class of pathological conditions. In **diabetes milletus** the percentage of sugar ranges from 0.5 to 8 per cent, and the specific gravity varies between 1.035 and 1.040. In **fevers** the increase of **urea** raises the density.

Acute and chronic parenchymatous nephritis are conspicuous examples of the latter variety. In these diseases the glomeruli are congested and degenerated, hence the amount of water secreted is lessened and the density of the urine correspondingly increased.

Specific gravity is decreased, on the other hand, by any condition which (a) reduces the amount of solids, or (b) increases the quantity of water secreted.

Interstitial nephritis (atrophied or contracted kidney) and amyloid kidney are instances of the former variety.

Diabetes insipidus furnishes a typical example of the latter class. In this disease the quantity of watery secretion is largely increased, but not the secretion of organic solids.

Hyperacidity is found in gout, lithiosis, and acute rheumatism, owing to the preponderance of uric acid; in fevers owing to the abundance of urea; and in diabetes because of the presence of acetons.

Alkalinity occurs in **cystitis, prostatitis, malignant**

diseases of urinary tract, **paralyses,** and any other condition which causes long retention and consequent fermentation of the urine. **Profuse hematuria** renders the urine alkaline.

Urea is the most important ingredient of normal urine, as it is also the most important of the nitrogenous excreta, which are practically all excreted in the urine. Urea is by far the largest of the end products of proteid digestion, and the quantity found in the urine practically determines the amount of proteids broken down in the body in a given time, the other forms of nitrogen found in the urine (creatin, ammonia, salts, and purin bodies) and those secreted by other channels (milk, sweat) being a negligible quantity. The precise rationale of its elaboration is unknown. It is assumed that the ammonium compounds resulting from proteid catabolism reach the liver through the portal system, are there converted into urea, and the latter eliminated by the kidneys. Suppression of liver function results in accumulation of ammonium compounds in the blood, and suppression of kidney function in accumulation of urea. There is evidence, however, that urea is formed to a limited extent by other tissues than the liver.

Increase of urea in the urine is the direct outcome of increased metabolism. Hence it is found in **fevers** and **inflammatory diseases,** in **diabetes, malaria,** and **pernicious anemia.**

Decrease of urea on the other hand points to abnormal decrease of metabolism, and is seen in **chronic nephritis, gout, rheumatism, malignant** and **constitutional diseases,** not so much as a result of these diseases, but as an accompaniment, due to the same metabolic disturbance as is causing the disease.

Uremia is sooner or later the upshot of a suppression of urea in the urine. Formerly it was thought to be due to the accumulation of urea in the blood (hence the name), but latterly it is agreed that the process of metabolism of which urea is normally the end-product is diverted, and produces abnormal toxins which are not eliminated by the kidneys, but circulate in the blood and poison the nerve centres.

Ureteral Function.—The urine which is being constantly formed in the kidney passes into the ureter, down which it is carried by the combined forces of gravity and peristalsis to the bladder, into which it is forced intermittently. The peristaltic contractions occur automatically every ten to twenty seconds, beginning at the pelvic opening and passing downward in the form of a wave. The ureter is therefore normally always filled with urine, each wave forcing out about 1-2 c.c. into the bladder.

Obstruction.—The ureter is not infrequently the seat of obstruction due to **impacted calculus, shreds of malignant growth, stricture, spasm,** or external

pressure from **pelvic** and **abdominal tumors.** The obstruction, of course, prevents the normal on-flow of urine and produces stasis in the renal pelvis **(hydronephrosis),** and if bilateral, eventual suppression of urine. Fortunately bilateral troubles of this kind are rare.

Infection to and from the bladder and kidney is occasionally carried by the ureters, causing pyelitis or cystitis as the case may be. Owing to the normal downward peristalsis, ascending infection is fortunately rare, and descending infection has, of course, not so prolific a starting place.

Innervation.—The nervous mechanism of the ureters is undetermined. Opinions waver between the view that it is a reflex phenomenon stimulated by the urine, and the theory of an automatic myogenesis similar to that of the heart muscle. Byron Robinson has recently pointed out that all of those functions which are under the domain of the sympathetic plexuses ("the abdominal and pelvic brain" of Robinson) exhibit a periodicity in the performance of their function, varying from once a month in the case of the menses to many times a minute in the case of the heart.

The bladder serves as a reservoir for the urine until it is voided from the body by the act of urination, being retained by the tonic contraction of the sphincter vesicae. It must be understood that the bladder, like the stomach, is always distended pre-

cisely in proportion to the amount of its contents, its muscular coat being in a constant state of tonic contraction around these contents, so that the bladder is really always "full." What is usually spoken of as a full bladder is really a fully distended bladder.

The urine, during its sojourn in the bladder, is practically a non-vital fluid and is liable to physical and chemical changes arising either from reactions within itself, or from pathological conditions of the bladder walls.

Decomposition and bacterial fermentation occur if from any cause the urine remains too long in the bladder, in which case it becomes alkaline (ammoniacal) and a source of toxic infection to the urinary tract. Such a phenomenon occurs in any condition which delays the voidance of urine, e. g., **enlarged prostate, vesical paralysis, malignancy, chronic cystitis,** and **brain diseases.**

Pyuria, pus in the urine, frequently results from suppurative and infectious conditions of the bladder. Epithelium, blood, and even nucleo-albumin may also be derived from broken-down conditions of the bladder-wall as in **malignancy, tuberculosis, calculus, cystitis.**

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Mechanism of Micturition.—The distension of the bladder by urine stimulates a reflex contraction of the muscular coat, and relaxation of the sphincter

vescicle, which would immediately empty the bladder were it not opposed by a voluntary contraction of the sphincter. As soon as the brain relinquishes this voluntary opposition the sphincter relaxes and the urine is voided by the vescical contraction, aided toward the end of the act by abdominal muscles.

The inhibitory conrol of the brain can only be maintained up to a certain point of stimulation, beyond which the spinal centre will act in spite of it. If the bladder distension should force a few drops of urine into the urethra the spinal stimulus becomes much more intense and proportionately difficult to inhibit, owing to the excessive sensitiveness of the urethral nerves. For the same reason, after urination has once begun, and urine is flowing through the urethra, it is very difficult to stop the act.

Inhibitory control of micturition is acquired; in infancy the act is purely reflex and involuntary.

Abnormal desire for urination both as to urgency and frequency, are seen in pathological conditions of the bladder and urethra.

Excessive desire, urgent and frequent, results from conditions which either (a) rapidly distend the bladder to its physiological limit, or (b) render the nerves so sensitive that an abnormally small distension stimulates the reflex.

The former class includes all those diseases which increase the secretion of urine, notably **diabetes, nephritis, cardiac hypertrophy,** and **neuroses.**

Of the latter class the chief conditions are those of **cystitis** and **urethritis**, in which the inflamed state of the membrane renders the nerves amenable to very slight stimulus. In these conditions a small degree of bladder distension excites the reflex contraction, squeezing a drop of urine into the highly sensitive inflamed urethra, and producing an overwhelming desire to micturate. Small quantities are of course passed at each act.

Subnormal desire, on the other hand, results from any conditions which (a) diminish urinary secretions, or (b) dull the sensibilities of the bladder or urethra.

In the first variety come all those diseases which diminish renal activity, notably **chronic nephritis, myocarditis, nephrosis**, and **neuroses**.

In the second list are classed **malignant diseases of bladder and urethra, tuberculosis**, and **chronic cystitis** and **urethritis**, in which the tissues are rendered lax and dull; and **central nervous lesions** which depress the general nervous activity. In any of these conditions an abnormally powerful distension is necessary to stimulate the reflex.

Over-concentration of the urine, hyperacidity, and other chemical conditions of the urine may also cause increased desire by unduly irritating the afferent nerves. This frequently occurs in **fevers, gout, rheumatism**, etc. It is probable, however, that these properly belong under the heading of **cystitis** and **urethritis**.

Innervation of micturition is mediated through the vescico spinal centre in the third lumbar segment, the afferent stimulus from the bladder being conveyed by the sensory fibres of the first to the fourth sacral spinal nerves, and the efferent impulse by the motor fibres of the same nerves. The sympathetic fibres passing out by the second to the fifth lumbar vertebrae (doubtless from the same centre) and reaching the bladder through the mesenteric ganglion, keep its muscular coat in a constant state of tone; and the hypogastric nerve plays a feeble part in its innervation.

Paralysis of the nervous tracts concerned in micturition, either by destruction of the spinal centre (most frequent) or by interruption of the afferent or efferent paths, produces paralysis of the act. In such cases the urine is not voided until distension become so great that it is forced out of the bladder by sheer mechanical pressure, after which it dribbles away involuntarily. It is rather an uncommon phenomenon, occurring chiefly in severe forms of spinal disease, e. g., **myelitis, sclerosis, late tabes dorsalis,** and **injuries.** It must be distinguished from

Involuntary micturition, which depends upon a suspension or interruption of the inhibitory influence of the brain. In this condition the urine is voided involuntarily, as soon as distension is sufficient to stimulate the bladder reflex, the bladder being completely and as a rule convulsively emptied at each

orgasm. This phenomenon occurs in **cerebral diseases, profound coma,** and **certain neuroses.** Abnormally deep sleep occasionally induces it (enuresis).

Psychic conditions may, by direct operation through the vesico-spinal centre, influence the act of micturition, either by suspending inhibition and precipitating the reflex (most frequent), or by inhibiting the reflex itself. Conspicuous instances of both these phenomena are frequently met with in cases of **psychic shock, hysteria,** and **melancholia.**

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The mechanism of defecation is similar to that of micturition. The feces are kept in the rectum by the contraction of the internal and external sphincters. When the rectum becomes sufficiently distended the distension stimulates a reflex contraction of the rectal muscles and relaxation of the internal sphincter by which the feces would be expelled but for the voluntary opposition of the external sphincter. When this opposition is inhibited by the brain, the external sphincter relaxes, and defecation takes place. In forced defecation (and it is usually more or less forced) the abdominal muscles assist in expelling the feces.

As in micturition, the control of the brain is possible only up to a certain degree of stimulation; beyond that point defecation occurs in spite of the will.

ease.

Abnormal desire for stool, both as to frequency and urgency, is seen in pathological conditions of the rectum and anus.

Excessive desire results from any conditions which (a) rapidly distend the bowel to stimulation point, (b) render the rectum unduly irritable so that an abnormally small distension (or even no distension at all) stimulates the reflex.

In diarrhea and dysentery both of these factors are usually active in producing frequent and urgent desire for stool. It must not be forgotten, also, that the liquid character of the feces makes a much more vigorous contraction of the sphincter necessary in order to keep them inside the rectum.

In proctitis the inflamed condition renders the rectum so sensitive to stimulus that the presence of small quantities of feces induces the reflex act of defecation with great urgency and pain (tenesmus).

Subnormal desire arises from any conditions which (a) reduce the quantity of feces reaching the rectum in a given time, or (b) render the rectum abnormally irresponsive to stimuli.

Constipation (diminished peristalsis) from any cause, of course, comes under the first head, while under the second may be classed **malignant diseases of the rectum, tuberculosis, chronic proctitis,** which render the rectal walls lax and dull, and central nervous lesions which depress general nervous activity. In any of these conditions considerable distension is necessary to stimulate the reflex, and in

many of them the pain attending defecation induces a voluntary retention.

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Innervation of defecation is mediated through an anal centre in the lumbar cord, the afferent stimulus being furnished by the sensory fibres and the efferent by the motor and inhibitory fibres of the sympathetics from the pelvic flexus and inferior mesenteric ganglia and the hypogastric nerve. That the phenomenon is under the influence of the higher centres is demonstrated by the effects of emotions and the involuntary defecation of infants.

Involuntary defecation results from an interruption of the inhibiting influence of the higher centres upon the sphincter. As soon as the distension of the rectum is sufficient to stimulate the reflex the feces are involuntarily voided by a vigorous contraction of the muscles concerned. This symptom occurs in **cerebral diseases, profound coma,** and certain **neuroses,** and very exceptionally in deep sleep. It must be differentiated from

Rectal paralysis, depending upon destruction of some part of the reflex tract concerned in the act of defecation. In these cases there is no defecation until the rectum becoms so full that the feces are forced out by physical pressure, slowly and with no effort. This is seen in severe forms of spinal disease as in **myelitis, sclerosis, late tabes dorsalis,** and **injuries** involving the lumbar region, and rarely in

neuritis by implication of the peripheral neuron concerned in the act.

Psychic influences may, by direct operation through the anal centre, modify the act of defecation either by suspending inhibition of the sphincter and inducing involuntary defecation (most frequent), or by inhibiting the reflex itself and retaining the feces. **Psychic shock, hysteria,** and **neurasthenia** furnish instances of both kinds.

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Feces.—As already stated, under digestion, very little absorption of anything but water takes place in the large intestine, and the alkaline reaction in this part of the tract favors bacterial putrefaction. By the time, therefore, the material reaches the rectum, under normal conditions, three consummations are reached; (a) the substance remaining contains only waste matter; (b), it has acquired a relatively solid consistency, and (c) it is in an advanced state of decomposition. It is then known as the feces, and is voided per anum.

The quantity of feces varies with the amount and nature of the food ingested, the average quantity being from 140 to 200 grams in twenty-four hours.

Abnormal Consistency.—Aside from the variations due to diet, the consistency of the feces varies directly with the length of time they remain in the large bowel. Under vigorous peristalsis (diarrhea) they pass very rapidly, there is no time for any great absorption of water, and the stools are usually

liquid. In diminished peristalsis (constipation), on the other hand, they remain in the bowel a long time, are inordinately drained of fluid, and are therefore dry and hard.

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The temperature of the feces, owing to fermentation, is higher than the body temperature—hence in taking rectal temperature care should be exercised not to insert the thermometer in a mass of feces.

Composition.—The constituents of the feces, of course, vary with the diet and other circumstances. Generally speaking, however, they contain the following ingredients:

Undigested food stuffs (principally fats).

Products of intestinal secretions (nitrogen).

Products of bacterial putrefaction (principally indol and skatol).

Bile salts and pigments (uro and starcobilin).

Inorganic salts.

The characteristic color of the feces is due to the bile-pigments; their odor to the skatol.

Lienteria, i. e., an abnormal quantity of undigested food in the feces, indicates, of course, that the alimentary tract is not properly disposing of the food ingested. It occurs in all digestive disorders. The particular stage of indigestion which these lienteria are found (chemically) may furnish information as to the precise part of the alimentary process at fault.

Mucus in the feces is indicative of **catarrh** when found in large amounts.

Clay-colored stools signify a lack of bile pigments and indicate a suppression or obstruction of the gall-bladder or common duct, as in **jaundice, cholecystitis, gall-stones, hepatic cancer,** etc.

Blood may come from any part of the intestinal tract and from many causes. **Hemorrhoids** are the commonest cause. **Enteritis, ulceration, cancer, tuberculosis,** are less frequent causes. In these cases the blood is usually comparatively bright red. Black blood (altered blood) originates in the upper alimentary tract, as from **duodenal,** or **gastric ulcer, swallowed blood, hepatic cirrhosis,** etc., and is partially digested during its passage.

Gall stones are often found in the feces. Their significance is obvious.

Microscopical and Chemical Contents.—Latterly Nothnagel, and still later Adolph Schmidt, have inaugurated a more thorough functional diagnostic of digestive and intestinal disorders by means of a minute and systematic examination of the feces, similar to the methods employed in gastric functional diagnosis. The system is, however, as yet rather too imperfect to enter into a practical text book.

Increased fat in the stools indicates (a) a deficient secretion or flow of bile, as in **jaundice, cholecystitis, hepatic cancer,** etc., (b) disturbance of pancreatic secretion, hindering fat digestion, as in **pancreatitis,** or (c) intestinal abnormalities interfering with absorption, as in **intestinal tuberculosis, malignancy, amyloid disease,** etc.

THE NERVOUS SYSTEM.

General.

The Neuron Doctrine.—The modern conception of the structure of the nervous system regards it as made up of a series of neurons. Each neuron consists of a nerve cell from which project a varying number of spoke-like processes, called dendrites; one of these processes is much longer than the rest, forming what was formerly known as the nerve trunk, and is called the axis cylinder, or axon.

The theory is that the nervous impulse originates in the metabolic changes produced by the stimulus in the nerve cell, and is propagated along the axon by a physico-electric current, always in the direction away from the cell. (This is known as the **polarity of the neurons.)** Reaching the end of the axon, the impulse is transmitted to the nerve cell of another neuron (through the latter's dendrites?) which, in its turn, is stimulated to metabolic changes and propagates a current along its axon. Thus the impulse reaches its destination by a relay process similar to that used in the long distance telephone.

Afferent and Efferent Neurons.—The neurons are arranged in two classes with reference to the direction of their impulses. In one class the polarity of the neuron is from the peripheries to the center of the system; these are called afferent or sensory neurons. In the other class the polarity of the neuron

is from the centers to the peripheries; these are called efferent, and usually motor, neurons.

Afferent and Efferent Impulses.—Afferent impulses, traveling from the peripheries toward the centers, include:

(1) **Sensory Impulses.**
- (a) Touch.
- (b) Pressure.
- (c) Temperature (heat and cold).
- (d) Muscle Sense (degree of muscle contraction).

(2) Special Sense Impulses.
- (a) Sight.
- (b) Hearing.
- (c) Taste.
- (d) Smell.

Efferent impulses traveling from the centers toward the peripheries, include:

(1) **Motor Impulses.**
- (a) General Muscle Motor.
- (b) Vaso-Motor (Muscles of Vessels).
- (c) Accelerator (Special Muscles).

(2) **Secretory Impulses.**
- (a) Gland Secretion.
- (b) Nourishment (Trophic).

Trophic Influence of the Cell, Wallerian Degeneration.—The cell exerts a nutritive or trophic influence upon the rest of the neuron, which appears to be in the nature of a current, for if the cell be cut off from the axon the axon degenerates on the

distal side of the cut-off, the degeneration proceeds along the fibre in the same direction as an impulse travel, viz., toward the end of the axon. A small amount of degeneration also occurs in the proximal end of the divided axon, but only in the immediate vicinity of the cut. Within a few hours of such division, histological changes also take place in the cell, from which it appears that the cell is also dependent in some measure on the axon for its integrity. Contrary to former views, we now know that this degeneration invariably follows division of a neuron and proceeds to completion before there is a reunion; it never heals by first intention.

Within a few days after division, regeneration begins, and proceeds in the same order as, and almost simultaneously with, degeneration. At the same time, restitution occurs in the elements of the cell. Within four to six weeks the axon is as fully regenerated as it is possible for it to be without connection being re-established with the cell, but it is never capable of functionating until this connection is re-established, either by a natural or a surgical uniting of the divided ends of the axon.

This process, after its investigator, Waller, is called Wallerian degeneration and regeneration.

Effects of Drugs and Physiological Conditions.— The irritability of both cells and axon is greatly modified by the action of certain drugs, and by physiological conditions.

The activity of the whole cerebro-spinal system seems to depend in a general way upon the activity of its highest part, the cerebrum. Physiological exaltations and depressions of the psychical faculties, such as are found in labor and sleep, joy and sadness, correspondingly exalt and depress the activity of the neurons throughout the system, increasing or diminishing their reaction to stimuli.

Delayed Conduction.—Pathological conditions of definite tracts and environs frequently increase the time of the passage of an impulse (delayed conduction). A conspicuous example of this is seen in **peripheral neuritis,** (inflammation of the lower sensory axons).

Certain drugs increase the activity of the cord and brain, chief among them being strychnia, ergot, nux vomica, belladonna and phosphorus; others depress these functions, notably opium, chloral, curare, bromides, and Indian hemp.

The Spinal Cord.

The spinal cord performs a double function. (1) That of a great conducting medium, its white matter consisting of long axons which pass upward to or downward from, the brain, and (2) That of a multiple center, its gray matter containing root and ganglion cells which play the part of subordinate brain centers in each segment of the cord.

The probabilities are that the central function of the cord is its oldest function, from the standpoint of evolution, each segment being originally a separate and independent center, and the joining of the segments and their long fiber connection with the brain a later development. The higher one goes in the scale of life the less independent a structure the cord becomes, the more important a part the brain plays, and the more numerous the conducting neurons between the two.

Afferent and Efferent Paths.—The cord receives the axoms of afferent neurons (originating in the skin, limbs, trunk, etc.), which enter it at different levels, and terminate in various manners. The impulses from these axons are transmitted to fresh neuron cells within the cord, whose axons either emerge from the cord again and carry the impulse back as an efferent impulse (reflex), or extend upward toward the brain and continue the impulse as an afferent one.

The cord receives the axons of efferent neurons (originating in the brain cells), which extend downward to various levels in the cord and terminate in various manners. The impulses of these axons are then transmitted to fresh axons within the cord, whose axons leave the cord auteriorly and terminate in muscles, etc.

Upper and Lower Neurons.—The afferent or efferent neuron between the periphery and the cord is called the lower neuron; that between the cord and the brain is called the upper neuron.

Posterior Spinal Nerves.—The axons of the lower afferent neurons enter the cord posteriorly, in symmetrical pairs, from either side of the body, one pair to each corded segment, and are called the spinal sensory nerves. The points at which they enter are called the posterior or sensory roots. There is a ganglion on each sensory root which appears to contain trophic cell elements of the lower neuron.

Sensory Paralysis, or **Anesthesia,** results from interruption in the course of these lower sensory neurons, or injury to the peripheral endings which paralysis is, of course, limited to the area supplied by the neurons involved. Such anesthesias are due to (1) traumatic injury to nerve-ends as from **burns, skin diseases,** etc., (2) vaso-motor disturbances at the peripheries, as **anemia** or **congestion,** and (3) lesions along the course of the axons, from **tumors, inflammation,** etc. **Peripheral** and **multiple neuritis** are conspicuous examples of this latter class.

Anterior Spinal Nerves and Roots.—The axons of the lower efferent neurons emerge from the cord anteriorly, in pairs similar to the afferent, and are called the spinal motor nerves. They terminate in and innervate skeletal muscles. Their points of emergence are called the anterior or motor root, and in these roots are located the trophic cell elements of the lower efferent neurons.

After emerging from the cord, and before leaving the vertebrae, the efferent axon of each segment coalesces with the afferent axon of the same segment, and travels between the cord and the periphery in the same sheath, both supplying the same peripheral area.

Motor Paralysis results from interruption in the course of these lower efferent neurons, or injury to their peripheral endings, limited to the area supplied by the interrupted neurons. Such paralyses are due to (1) traumatic injury to nerve ends, as from **burns, skin diseases,** etc.; (2) lesions in the course of the axons, as **tumors, inflammations,** etc.

Owing to the fact that the lower motor and sensory neurons travel in the same sheath, lesions of these lower neurons almost always involve both, and hence produce motor and sensory paralysis. A double paralysis of a limited area is therefore suggestive of a lesion of the lower neuron.

Atrophy.—In diseases of the nerves where the lesion is situated between the root-cells and the peri-

phery, or in which the root cells themselves are involved, there is rapid atrophy of the muscles concerned in addition to paralysis. Conversely where rapid atrophy is associated with paresis, the lesion may be safely located in one of these positions.

In **poliomyelitis** there is atrophy because the anterior horns are themselves inflamed and later degenerated.

In **diffuse myelitis** the same is true.

In **progressive muscular atrophy,** the degeneration begins in the lower neuron, often in the muscle, hence atrophy precedes paresis.

In **lateral amyotrophic sclerosis,** the degeneration begins in the spinal tracts and later attacks the root cells, hence paresis precedes atrophy.

In **multiple neuritis** the lower neurons are inflamed, hence there is atrophy if the inflammation continues for any length of time.

In diseases of the cord which involve only the tracts, and in purely cerebral diseases, there is no such atrophy, because there is no interruption of the course between the limb and the root-cell, the lesion being above the root-cell.

Conducting Paths in the Cord.—The conducting neurons of the cord have been demonstrated to pursue more or less definitely marked paths or tracts, according to the impulses they convey. These tracts are more definite in the case of the descending (efferent) than of the ascending (afferent) neurons.

Ascending Tracts.—The most important ascending, or afferent, tracts and their location in the cord, are:

(1) Tract of Goll, in the posterior median column.

(2) Tract of Burdoch, in the posterior intermed. column.

(3) Flechsig's Tract (Direct Cerebellar), in the lateral column.

(4) Gowers' Tract (Ventral Cerebellar), in the lateral column anterior to Flechsig's Tract.

Course of Posterior Fibres.—Contrary to former ideas, the sensory fibres which enter the cord at the posterior roots (vide supra) do not all pass up the posterior columns of the cord. Indeed very few do so. The great majority of them penetrate the gray matter of the cord and transmit their impulses to tract cells, whose axons pass up the lateral tracts (Flechsig and Gowers), or simply pass over to other parts of the cord, or communicate with an anterior motor neuron, forming a reflex arc.

Of those fibres which do pass up the posterior columns, some continue to the medulla; others often passing upward for a short distance branch off and terminate in the tract of Flechsig. At these respective terminations they transmit their impulse to new neurons, which carry them to the brain.

Muscle Sense.—Experiment and observation show that the posterior and lateral tracts do not play any important part in the transmission of ordinary sen-

sory impulses (touch, pressure, etc.), but conduct principally impulses of muscle sense, or those impulses which acquaint us with the position and degree of contraction of our muscles. The great bulk of these impulses are conveyed to the cerebellum, which is the chief functionating center for co-ordination.

Inco-ordination.—Anything which interrupts the passage of these muscle sense impulses deprives the the cerebellum of information which is essential to the performances of its co-ordinating function, and results in inco-ordination, manifested clinically by inability to accomplish purposeful movements of the parts involved, staggering gait, loss of balance, inability to perform delicate tasks with hands, etc.

Conversely, these clinical symptoms always point to interruption of muscle sense impulses. Whether the interruption is in the lower or upper neuron must be determined by other symptoms.

In **myelitis** the spinal tracts, and in **neuritis** the peripheral sensory neurons, are inflamed, and cannot transmit impulses; hence ataxia results.

In **Locomotor Ataxia** the posterior columns (Goll and Burdoch) are degenerated; hence the muscle sense can no longer be transmitted along these tracts and ataxia results.

Lateral Scleroses, by virtue of the degeneration of the lateral tracts (Flechsig and Gowers) produce a similar result.

Touch, Pressure, Temperature.—Of the tracts by which impulses of touch, pressure, temperature, etc., are conveyed, nothing is definitely known. Experiment and observation demonstrate that the different impulses are conveyed by separate fibres (dissociated sensations) and make it probable that there are many different paths by which they travel.

True sensory paralysis is rare—complete anesthesia still rarer. When it does occur it is mostly peripheral in origin as already described.

Given the integrity of the sensory peripheries, however, it is rarely that the sensory impulses cannot find some path or other by which to reach the brain. Interruption of any of the ascending tracts of the cord never produces more than partial paralysis, except in rare cases of growths or degenerations which involve the whole section of the cord, in which case, of course, both motor and sensory paralysis are complete. Spinal anesthesias are usually bilateral.

Anesthesias due to cerebral lesions, hemorrhage, tumors, softening, etc., are extremely rare, difficult to demonstrate, and never intense. They are usually unilateral.

Syringomyelia is the disease which affords the most interesting manifestations of sensory conduction. The anatomic basis of this disease is the formation of cavities in the substance of the cord, and it is clinically attended by some very peculiar and

characteristic sensory phenomena, chief among which is a discriminate insensibility to the various stimuli of temperature, pressure and touch (dissociation of sensations).

A far more common sensory phenomenon is that of irritation, due to primary inflammation of either the lower or upper sensory neurons, and manifested clinically by pains referred to the periphery of the affected neuron. These pains are seen in **locomotor ataxia** (degeneration of the posterior column) **multiple neuritis** (inflammation of lower neurons, **myelitis** (inflammation of the spinal tracts), and other similar diseases, in their early stages; on supervention of degeneration or compression they give way to partial anesthesia.

Decussation of Ascending Tracts.—The sensory neurons which traverse the tract of Gowers decussate or cross at the medulla and go to opposite sides of the brain. The other tracts are mostly continued to the brain in a direct course. This, however, only applies to decussation or directness **en masse,** for there is partial crossing of sensory fibres all the way up the cord.

Physiologically it is, of course, certain that the sensory impulse from the body peripheries are received on opposite sides of the brain, and inasmuch as the brain is a higher type of reflex arc, this is in accordance with what we know of the crossed location of the motor centres. However, this crossing

of the sensory tracts has no clinical significance, because the crossed reception of sensory impulses is impossible to demonstrate, the sensorium of the brain always referring them to the side on which they originate.

Descending Tracts.—The principal efferent or motor tracts in the cord, and their topography, are:

(1) Direct Pyramidal Tract (anterior motor) in the anterior columns.

(2) Crossed Pyramidal Tract (lateral motor) in the lateral columns.

Course of Descending Tracts.—The course of the motor tracts is much more definitely traced and better understood than that of the sensory tracts. The exoms forming the motor paths are received from the brain, through the medulla, and continue their course to the various levels in the cord, where they communicate their impulse to a tract cell whose axon passes out of the cord by an anterior root, and terminates in a group of skeletal muscles.

Motor Paralysis.—Any interruption in the course of these tracts produces motor paralysis in the part below the interruption. A careful examination of the muscles involved in such paralysis should therefore enable the diagostician to locate the level of the lesion; and so it usually does to a certain extent, but unfortunately not with anything like the definiteness that the data would lead one to expect. In fact, actual spinal paralyses are not nearly so sharply defined as hysterical paralyses.

Poliomyelitis (inflammation of the anterior horns), **progressive muscular atrophy,** which later attacks the anterior root cells, and **amyotrophic lateral sclerosis** (involving the crossed pyramidal tracts) all produce motor paralysis by interrupting the motor tracts of the cord. In **multiple sclerosis** there is seldom true motor paralysis because in spite of the diffuse nodules the neuraxons usually persist and functionate.

Myelitis, which inflames all the tracts, produces motor paresis.

Decussation of Descending Tracts.—The lateral motor tracts decussate in the medulla and supply opposite sides of the body. The anterior motor tracts continue their course directly down the same side of the cord, but eventually their fibres cross at various levels in the cord.

Crossed Paralysis.—Injury to a motor tract above the point of discussation will produce a crossed paralysis, i. e., a paralysis of the opposite side of the body to the lesion, but on the same side of the head and neck, as the cranial nerves are given off before decussation takes place. In case the cranial nerves are not injured (a rare combination), there is no way of diagnosing the crossed nature of the paralysis.

Injury to a motor tract in the lateral column of the cord will produce a paralysis on the same side below the seat of the lesion.

Injury to the direct motor tract will give paralysis on both sides. This class of paralysis is frequently associated with lesions of the lateral sensory tract, involving ataxia.

As a matter of practice, however, paralysis due to lesions in the spinal tracts are almost invariably bilateral (paraplegia) because the pathological process usually involves both sides of the cord.

The Cord as a Center.—The principal function of the cord as a multiple center is that of producing true reflexes, each segment of the cord functionating as an arc for reflexes produced by its own sensory nerve. The sensory impulse is usually communicated to several motor neurons, and sent back as a multiple efferent impulse, innervating a group of muscles in a purposeful manner (co-ordinated reflex).

Overflow Reflex.—Sometimes the stimulus is so vigoruous that the sensory impulse, reaching the cord, is communicated to motor neurons quite outside the purposeful group and produces motion in an unnecessary group of muscles. This is called a reflex overflow.

Special Reflexes.—In the accompanying table will be found a list of the more important spinal reflexes, the afferent and efferent paths, and the location of the centres concerned in them.

REFLEX.	TECHNIQUE.	SPINAL SEGMENT.
Biceps Reflex.	Tapping Tendons produces Flexion of Forearm	Fifth and Sixth Cervical.
Triceps Reflex.	Tapping Tendons produces Extension of Forearm	Sixth Cervical.
Palmar Reflex.	Stroking Palm causes Closure of Fingers...	Eighth Cervical to First Dorsal.
Abdominal Reflex.....	Stroking Side of Abdomen causes Retraction	Ninth to Twelfth Dorsal.
Cremasteric Reflex.....	Stroking Inner Side of Thigh causes Retraction of Scrotum	First and Second Lumbar.
Patella Reflex.	Striking Tendon at Knee causes Extension of Leg	Second and Third Lumbar.
Achilles Reflex	Tapping Achilles Tendon causes Flexion of Ankle	First to Third Sacral.
Ankle Clonus.	Extension of Achilles Tendon causes Flexion of Ankle	First to Third Sacral.
Plantar Reflex.	Tickling Sole of Feet causes Flexion of Toes, or Extension of the Great Toe and Flexion of others	First to Third Sacral.

Absence of a reflex may indicate an interruption in either of the lower neurons in the afferent or efferent, or a destruction of the spinal are in the cord. The former cause may be excluded by absence of other indication, such as flabbiness and atrophy of muscles. Far the greater proportion of reflex failures are due to injury to the spinal arc.

Naturally the reflexes whose arcs are lowest down in the cord are the most commonly utilized in diagnosis, as they indicate the condition of the cord above the arc. Of these low-down reflexes the knee-jerk (patellar) and tendon Achilles are the most easily elicited and constant.

The normal knee-jerk indicates integrity of both afferent and efferent (lower) neuron of the third lumbar nerve, of the commisural arc at the third lumbar segment, and of the tracts from the third lumbar segment up to the brain. Thus a normal knee-jerk practically assures integrity of the entire spinal tract.

Exaggeration of the knee-jerk indicates an interruption in one or more of the descending spinal tracts between the third lumbar segment and the brain, cutting off the inhibitory influence of the brain. For the same reason an exaggerated reflex is associated with hypertonus of the muscles. This condition is seen in **multiple sclerosis,** where the tracts are studded with nodules, **amytrophic lateral sclerosis,** in which the lateral motor tracts are

degenerated (very marked), in **cerebellar disease,** where the co-ordinating influence upon the tendons is cut off, and in all cerebral lesions. **Central myelitis** gives this symptom, because it involves the upper and not the lower neurons.

Absence of Diminution of the knee-jerk indicate interruption in the afferent or efferent neuron of the third lumbar nerve, or injury to the commissural arc, or disease of the posterior tract of the cord which includes destruction of the arc. The knee-jerk is abolished or lessened in **neuritis** and **progressive muscular atrophy** because of the inflammation of the lower neurons; in **locomotor ataxia** because of degeneration in the posterior tract and consequent destruction of the spinal arc. In the latter disease a careful examination of the various reflexes whose arcs are at different levels will indicate the upward limit to which the degenerative process extends. In **diffuse myelitis** the same occurs because the roots of the lower neurons are involved.

Both exaggeration and diminution of reflexes may be due to exalted or depressed conditions of the cord, caused by drugs or mental emotions, but these indications are as a rule easily recognized or excluded.

The tendo Achillis and plantar reflexes of course give information concerning the spinal tracts, from a still lower level, and these and other reflexes exhibit certain peculiarities and give information concerning their own specific neurons.

Tonic Functions of Cord.—Another central function of the cord is the furnishing of tonicity to the muscles. The cord is continually sending out a steady stream of nerve energy to the muscles which keeps them in a constant condition of partial contraction, or "tone," so that when they are needed, voluntary innervation will not have to overcome complete inertia before they can be moved.

This outflow of tonic energy is controlled and modified in an important manner by the brain, by means of inhibitory currents.

Differentiation of Spinal and Cerebral Paralysis.—Interruption of the course between the brain and the spinal neuron, in other words, injury to or division of the upper neuron, interferes with the inhibitory influence of the brain upon the tonic function of the cord, and an excess of tonic energy is poured out by the latter into the muscles. This is manifested clinically by a spastic condition of the muscles involved, and an exaggeration of reflex.

Interruption of the course between cord and periphery, i. e., injury to or division of the lower neuron, results in a cutting off of the tonic current, manifested clinically by a flaccid atonic condition of muscles involved and absence of reflex.

Conversely, therefore spastic paralyses and exaggerated reflex indicate injury to the upper neuron or brain. Flaccid paralyses and absence of reflex point to injury to the lower neuron. This should be

one of the first diagnostic points to be determined in the investigation of a paralytic condition.

In **peripheral neuritis** (inflammation of the lower neuraxon), **poliomyelitis** (inflammation of anterior roots), and **progressive muscular atrophy** (degeneration of the lower neuron), the tonal path is interrupted, and these diseases are characterized by flaccidity of the muscles involved, and diminished reflexes.

In **lateral amyotrophic sclerosis,** there is first spasticity and exaggerated reflexes, as long as the process is confined to the lateral tracts, followed by flaccidity and diminished reflexes when the root cells are attacked.

In **multiple sclerosis,** involving only the spinal tracts, and in all cerebral paralyses, the muscles are spastic and the reflexes are exaggerated. In **central myelitis** there is spasticity, because the upper neurons only are involved; in **diffuse myelitis** the reverse, because the roots of the lower neurons are inflamed.

Spasticity manifests itself in completely paralyzed limbs by resistance to passive motion. In partially paralyzed limbs it is shown by the jerky character of the movements; in the legs by a peculiar dragging walk (spastic gait).

THE MEDULLA OBLONGATA.

The medulla oblongata occupies, both anatomically and physiologically, a midway position between the cord and the brain. Like the cord, it performs the double role of a conducting medium for axons and a center for the governance of certain functions.

The direct fibres of the posterior column terminate in the medulla and their impulses are here transmitted to fresh neuron cells whose axons pass upward to the cerebrum and cerebellum. The lateral afferent fibres discussate in the medulla to pass to opposite sides of the brain.

The efferent or motor fibres, descending from the brain pass through the anterior portion of the medulla (pyramid) and pass downward into the cord, a portion of them decussating and some continuing in a direct course.

The Medulla as a Center.—The central canal of the cord widens out in the medulla and forms the floor of the fourth ventricle, the roof of which is formed by the base of the brain. From this chamber originate all the cranial nerves but the olfactory and the optic. All of these nerves but one (the pneumogastric) innervate the region of the head and neck.

Bulbar Centres.—In addition to the cranial nerve origins, the medulla contains certain important centres which, while they are not usually regarded as reflex centres, undoubtedly functionate as true co-

ordinate reflex arcs, the afferent stimulation being automatically furnished by the organs which they innervate. In many of these bulbar reflexes the cranial nerves take part.

These bulbar centres and their functions are as follows:

(1) Respiratory. (4) Deglutition.
(2) Vaso-Motor. (5) Vomiting.
(3) Cardio-Inhibitory. (6) Heat Regulation.

The Respiratory Center controls the muscular function of respiration by means of neurons which pass down the cord terminating at various levels and communicating their impulses to the lower motor neurons which supply the muscles of respiration, notably to the phrenic nerves at the fourth and fifth cervical segments, special accessory, and the intercostals.

Automacy of Respiration.—Many authors ascribe to the respiratory center an automatic character similar to that usually ascribed to the heart muscles, and many ingenious experiments have been performed to demonstrate the hypothesis. However, the point must be regarded for the present as unsettled. From a deductive standpoint the writer is inclined to the reflex explanation in spite of the apparent predominance of contrary evidence.

Bulbar Paralysis.—Injury to or disease of the medulla, if it involves, as it usually does, the respiratory center, of course paralyzes respiration and

causes death. Dislocation of the atlas as a rule produces instant death by sudden trauma and compression of the medulla.

In amyotrophic lateral sclerosis the atrophy eventually reaches the medulla and ends the scene of paralyzing either respiration or deglutition. **Multiple neuritis** frequently has the same outcome from inflammatory investment of the lower neurons concerned in respiration.

* * *

Vaso-Motor Center, Vaso-Constrictor Nerves.—The vaso-motor center controls the constriction and dilatation of the vessel by means of the vaso-motor nerves, whose lower neurons pass out of the cord at the anterior roots. These neurons are called vaso-constrictor nerves; this stimulation causes contraction of the muscles of the vessels, and a consequent rise in blood pressure. The center performs for these muscles the same tonic function that the cord centers perform for the skeletal muscles, and maintains the level of blood pressure necessary to life. Interruption of any of the neurons causes dilation and a fall in blood pressure in the vessel supplied; destruction of the center causes a general dilatation and fall of pressure, resulting in death.

Vaso-Dilator Nerves.—It is an experimental fact that neurons exist whose stimulation causes dilatation of the vessels, and it is usual, therefore, to speak of these as vaso-dilator nerves. Inasmuch, however, as these neurons are shown to have no reflex center,

as the constrictors have, and considering further, that physiology furnishes no precedent for the assumption of a nerve-current which relaxes a muscle, it would seem far more reasonable that the so-called vaso-dilator nerves are afferent neurons which set in motion an inhibitory reflex, restraining the function of the constrictors, in the same way that muscle sense impulses inhibit the innervation of antagonistic muscles and relax one or the other.

THE PONS VAROLII.

The Pons Varolii.—The pons, as its name implies, is a connecting link between the various parts of the brain, i. e., between the two cerebral hemispheres, between them and the cerebrum, and between all three and the medulla. Its function, so far as is known, is entirely that of conducting impulse from one to the other, and furnishing passage to the great sensory and motor tracts.

THE CEREBELLUM.

The Cerebellum.—The function of the cerebellum is at the present time a subject of much discussion. The preponderance of opinion, however, ascribes to it the office of co-ordinating motor impulses, i. e., directing them to a purposeful and economic end. This is, of course, stating its function in terms of its net result, for in order to accomplish this end it must also take cognizance of sensory impulses. It would

therefore be better to say that the function of the cerebellum is to strike a balance between the nervous income and expenditure, to the most purposeful and economical utilization of nervous capital.

Information of the Cerebellum.—It is necessary that the cerebellum be kept constantly informed of every item of nervous income and expenditure. Of the income it receives information by way of the sensory tracts, principally from the muscles (muscle sense) but partly also from all other peripheries. Of the expenditure it receives advice by means of commissural fibres from the cerebrum, by fibres from the motor tract as the latter passes through the pons, and possibly by the recurrent fibres which re-enter the cord from the lower motor neurons just after they leave the roots.

The paramount sources from which the cerebellum receives afferent impulses are:

(1) The skin,

(2) The muscles,

(3) The semi-circular canals of the ear. These canals are constructed on three planes, at right angles to each other; they communicate freely, and are filled with endolymph, which by its outward pressure on the hair follicles lining the canals transmits sensations to the cerebrum and cerebellum. The least deviation of the head in any plane disturbs the center of gravity in the canals, a sense of which is immediately transmitted to the cerebellum. In this way the canals exercise an important influence in the maintenance of static, and probably also of dynamic equilibrium.

Inco-ordination.—Interruption of the passage of sensory impulses from any of these sources to the cerebellum, or incapacity on the part of the cerebellum to receive them, produces inco-ordination, manifested clinically by impairment of purposeful movements of the parts involved. The interruption may occur either in the lower or upper neuron, or the cerebellum itself may be the seat of the lesion. Its location must be determined by considering accompanying symptoms.

Peripheral neuritis produces ataxia by interrupting the transmission of impulses from the skin and muscles at the periphery.

Locotomor Ataxy manifests ataxia because of injury to the posterior columns of the cord—one of the paths of the upper sensory neurons (vide supra).

In **multiple sclerosis** the nodules are scattered through the tracts, effecting a partial and diffuse interruption of sensory impulses, the result being a partial form of ataxaia which is now understood to be the explanation of intention tremor.

In **lateral amyotrophic sclerosis,** on the contrary, the process is always sharply limited and the cerebellar tract left intact; hence, amid all the havoc wrought by this formidable disease, co-ordination is

Myelitis, in which all the tracts are involved, gives ataxia, but the motor paralysis masks it.

In a hereditary form of locomotor ataxia, known as **Friedreich's Disease,** the inco-ordination extends to the muscles of speech, and the muscles of the

trunk are involved, making it difficult to maintain static as well as dynamic equilibrium.

That the sense of vision plays an important part in co-ordination is proved by the fact that in all the ataxia diseases referred to the inco-ordination becomes more marked when the eyes are closed (Romberg's symptom).

Mechanism of Co-ordination.—By just what means the cerebellum exerts its co-ordinating influence on the motor impulses is not known. Possibly by impulses sent to the motor area of the cerebrum which instruct the latter in the distribution of its motor currents; possibly by inhibitory impulses given to the motor fibres on their way down through the pons; possibly by direct neurons of its own to the muscles concerned (comma tract?).

Degeneration or atrophy of the cerebellum produces complete ataxia, including the facial muscles. This condition is seen in a rare disease known as hereditary cerebellar ataxia. Motor power is intact. Muscular sense and general sensibility are unimpaired, as are also the cutaneous reflexes; but the tendon reflexes are exaggerated because the tendons are under the influence of the cerebellum. The facial movements are noticeably purposeless and absurd.

Relation of Cerebrum to Cerebellum.—It may be said that in removal or injury of the cerebellum, although it produces temporary ataxia and inco-ordi-

nation, without any impairment of motor power, the ataxia and inco-ordination in man, are largely regained after a time, provided the cerebrum is intact. This physiological fact coincides with the anatomical fact that a goodly proportion of neurons which convey muscle-sense are distributed to the cerebrum, and affords another instance of the dominant capacity of the cerebrum, which is able to assume a great many functions in the absence of their customary centres.

A notable exception to this is found in the case of the disease just mentioned, hereditary cerebellar ataxia. In this trouble the cerebrum does not compensate for the cerebellum, doubtless because there has been no conscious education in co-ordination, and the cerebrum therefore has no experience to fall back upon.

THE CEREBRUM.

The cerebrum is, both topographically and functionally, the highest part of the nervous system. Its function is but little understood, its activity being dependent upon consciousness—that unknown and unknowable factor in the physiologic equation. We are only able to state, in a general way, that it is the seat of intelligent and voluntary function. Moreover, it seems that it only becomes so after repeated impressions and experiments, for the brain of an infant can hardly be regarded as a seat of intelligence or volition; and this trial and error process (even the passive part of it) is only possible in the presence of consciousness, for no impression can be made on the cerebrum during unconsciousness.

Psychical Functions.—Of the higher operations of the brain, commonly called psychical, including the phenomenon of consciousness, nothing can be predicated by physiology. They are generally supposed to be functionated in the outermost layer of the cortex (molecular layer), the axoms of whose neurons do not pass out of that layer, but communicate freely and completely with each other. This, however, is a purely histological hypothesis.

Cortical Region.—The pyramidal and polymorphous layers of the cerebrum, consisting chiefly of gray matter (non-medullated), send axons both upward into the molecular layer and downward into the medulla of the brain.

The Cerebral Medulla.—The medulla of the brain consists almost entirely of white matter (medullated), representing axons from neuron-cells in the upper layers and in the spinal cord. The fibers composing the medulla are classified according to their anatomy and function.

(1) **Association Fibers,** connecting one part of the cortex with another.

(2) **Commissural Fibers,** connecting the two hemispheres, cerebellum, and pons.

(3) **Projection Fibers,** which come from or go to the medulla oblongata and cord, and from the great sensory and motor tracts. These fibers, after leaving the cortex, sweep backward, in the form of an arc (corona radiata), getting closer together the lower they are traced, and pass in a very compact bundle on each side, through the capsule (the motor fiber through the auterior and for part of the posterior limb, the sensory through the back part of the posterior limb) down to the medulla oblongata, where they decussate, and into the cord.

Cerebral Motor Paralysis.—Trauma or disease of the great sensory and motor tracts during their passage through the capsule and pons, where the fibers are compressed into a very small sectional area, naturally includes all of the neurons in the injury, and produces a paralysis of all the parts supplied by the tract—partial or complete according to the extent of the injury or compression. If the lesion is above the

cranial nerve roots (as it usually is), the result is a crossed paralysis involving the same side of the head and neck and the opposite side of the body. If it is below the cranial nerve nuclei, the paralysis involves the opposite side of the body.

Hemorrhages into the capsule are very common, as are also **embolisms** of the middle cerebral artery, **tumors, gummata** and **abscesses** involving the capsule. The first two lesions usually cause a sudden and complete paralysis; the other three lesions usually produce gradually progressive hemiplegia.

* * *

Cerebral Localization.—Experiment and observation have demonstrated that certain areas of the cortex, being stimulated, functionate various parts of the body; one area, for example, always moving the leg, another the arm; and still smaller points are found in these areas which influence detailed sections of the parts in question, as the foot and toes, hands and fingers. The same is true of special functions, such as speech, hearing, etc. Hence we conclude that every part of the body is represented, afferently and efferently, by a certain definite area in the cortex, although the sensory areas, naturally, cannot be mapped out with the definiteness of the motor areas.

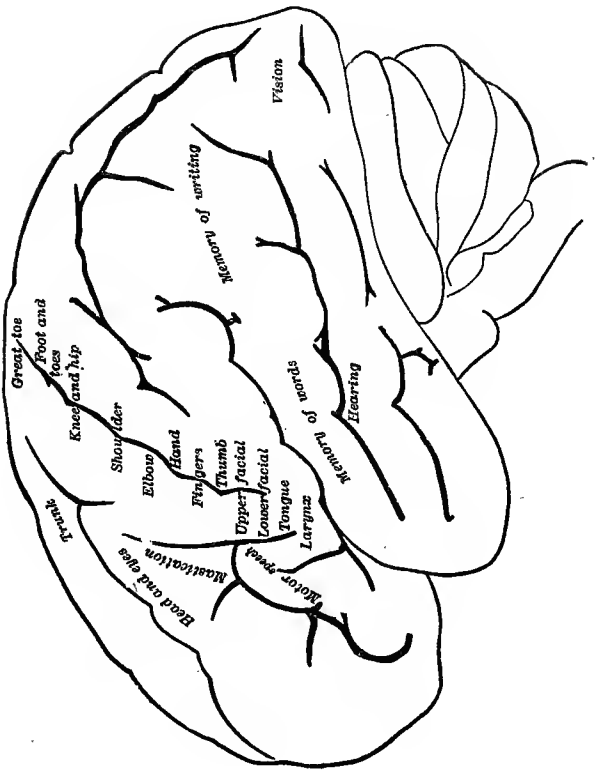
Monoplegias.—Inasmuch as the motor centres of the cortex are separated from each other by a more or less measurable distance, traumata and degener-

ative processes of the cortex, unless extraordinarily extensive, do not involve more than one or two of these centres. Hence the paralyses produced by cortical lesions do not as a rule involve more than one group of muscles, and are known as monoplegias. Conversely a monoplegia may as a rule be diagnosed as due to a cortical lesion, for in no other part of the cerebro-spinal course are peripheral groups represented by neurons sufficiently isolated to incur separate injury.

In certain types of **cortical epilepsy** the central irritation starts in one of these cortical centres, and the fit begins with a corresponding disturbance in the group of muscles controlled by that centre (Jacksonian Epilepsy).

Location of Motor Areas.—The motor areas are for the most part situated along the fissures of Rolando and sylvius, and a description of the probable location of the centres for the most important parts of the body will be found in the accompanying table. The location of special senses centres will be indicated in discussing those senses:

It will be readily seen from the table and illustration that the arrangement of the centres facilitates the occurrence of monoplegias. These are quite frequent in practice and it is usually a simple matter to diagnose the exact spot in the brain at which the lesion exists. Frequently, too, the lesion will include two adjoining centres, such as those of the face and


Great toe
Foot and toes
Knee and hip
Shoulder
Elbow
Hand
Fingers
Thumb
Upper facial
Lower facial
Tongue
Larynx
Trunk
Head and eyes
Mastication
Motor speech
Memory of words
Hearing
Memory of writing
Vision

arm, or of the arm and leg, and a paralysis of these two regions is then produced which is called an associated plegia. But it is apparent that an associated cortical paralysis of the leg and face, without paralysis of the arm cannot occur as the result of a single cortical lesion.

The greatest care must be exercised in differentiating between the various centres concerned in the functions of speech, hearing, and writing, and the effects of injury to these foci. Each of these functions is made up of two distinct operations of the brain, performed by two distinct and separate centres, which may be classified as follows:

Hearing.—(1) Perception of sound; (2) Storing of Sounds (auditory memory).

Sight.—(1) Perception of images; (2) Storing of Images (visual memory).

Speech.—(1) Recognition of stored sounds (spoken words); (2) Motor Speech.

Writing.—(1) Recognition of Stored Images (written words); (2) Motor Writing.

It will be readily apparent that the function of speech depends upon that of hearing since sounds cannot be reproduced which have not first been perceived and memorized. In like manner the function of writing depends upon that of sight, since the images cannot be reproduced which have not been first perceived and memorized. Psychologically the acts of storing and reproducing an impression are

identical ("there can be no impression apart from expression").

It will be further seen that the centres for these intradependent functions are situated immediately above each other in the brain as follows:

Motor Speech,
Memory of Spoken Words,
Hearing,
Arm and Hand,
Memory of Written Words,
Vision.

Pure Deafness.—A lesion of the auditory centre, or centre of pure hearing, produces inability to perceive sound. The current conveyed by the auditory nerve produces no effect upon it. This lesion is not necessarily fatal to speech, in these days of modern resource and patience, as the patient may be taught by sight, and, in the absence of sight, even by thought, to recognize and reproduce motions of the mouth and tongue. This form of deafness is called pure deafness.

Word Deafness.—A lesion of the centre of sound memory produces inability to store sounds, and therefore to recall them. This form of deafness is called word deafness, and is fatal to intelligent speech, because the word desired to be spoken cannot be thought of. In regard to speech this is known as amnesic aphasia.

Pure Blindness.—A lesion of the visual centre

produces inability to perceive an image. The current conveyed by the optic nerve makes no impression upon it. This form of blindness is called pure blindness and can be overcome by patient education so far as the ability to write is concerned.

Word Blindness.—A lesion of the visual memory centre produces inability to store and hence to recall images. This form of blindness is called word blindness and is fatal to intelligent writing because the word desired to be written cannot be recalled. In regard to writing this is called amnesic agraphia.

Aphasia.—The form of aphasia dependent upon injury to the sound memory centre (amnesic aphasia) has already been described. The other form of aphasia is that which results from injury to the motor mechanism of speech and is called motor or pure aphasia.

Agraphia.—The form of agraphia resulting from injury to the centre of visual memory (amnesic agraphia), has been described. That form which comes of injury to the motor mechanism of the arm and hand is called motor or pure agraphia.

Soul Blindness and Deafness.—There is still another form of word blindness and deafness in which sounds are heard and images seen, and both are stored and reproduced, but no connection is perceived between the sound or the image and any conception of the mind. This is known as soul blindness and deafness. It depends on no special centre, but upon a general dissociation of the cerebral areas.

REACTION OF DEGENERATION.

Reaction of Degeneration.—A similar process of degeneration occurs in a neuron which is cut off from its cell by pathological lesions in the intervening axon, such as new growths, sclerosis, inflammatory congestions, etc. The process is then known as secondary degeneration, ascending or descending as the case may be, and is clinically detected and measured by certain abnormal ways in which the neuron and the muscles it supplies react to electrical currents. This is known as the Reaction of Degeneration and may be briefly stated as follows:

In health, both nerve and muscle react to the faradic current by contracting sharply upon application of the needle, and to the galvanic current by a contraction at the closing and opening of the circuit, but not during the passage of the current, the Kathode contraction being more vigorous than the anode.

In complete reaction of degeneration neither nerve nor muscle reacts to faradic current; the nerve does not react to galvanic; the muscle contracts slowly and undulatingly to the galvanic, and exhibits as good a reaction to the kathode as to the anode.

In incomplete reaction of degeneration the reactions exhibit intermediate between normal and complete degeneration.

Reaction of degeneration enables us to locate the lesion in the lower neuron or in the root-cells, i. e.,

between the periphery and the trophic ganglia; and the persistence or improvement of the abnormal reaction indicates a grave or favorable prognosis of the restitution of the integrity of the neuron.

Reaction of degeneration is generally associated with muscular atrophy, because the trophic influence of the ganglion is also exerted upon the nutrition of the muscle.

It does not occur in diseases involving only the cord tracts or upper neurons, or in cerebral lesion. **Multiple neuritis,** involving inflammation of the lower neuraxons; **poliomyelitis,** inflammation of the anterior horns; and **Progressive muscular atrophy,** involving both lower neuraxons and anterior roots; all give reaction of degeneration. **Amyotrophic lateral sclerosis** gives reaction of degeneration in its later stages, when it attacks the motor roots.

SPECIAL FUNCTIONS.

Vision.

Ocular Movements are performed by means of the extrinsic muscles of the eye, the movements executed by each pair being as follows: Superior Rectus, upward and inward; Inferior Rectus, downward and inward; External Rectus, outward on vertical axis; Internal Rectus, inward on vertical axis; Superior Oblique, outward on antero-posterior axis; Inferior Oblique, correcting the inward deviation of the recti.

These muscles are in a constant condition of tonicity and oppose each other.

Muscular Imbalance is the result of inadequacy on the part of one of the extrinsic muscles, causing a deviation of the eyeball in the direction of the opposing muscle or muscles. This condition may be overcome for a time by excessive innervation of the faulty muscles, but eventually this gives way, and strabismus ensues. The inadequate muscle is commonly one of the recti, since these bear the brunt of the work. Inadequacy of the **external rectus** causes **convergent strabismus**; **internal rectus** causes **divergent strabismus.**

Muscular Innervation.—All the extrinsic muscles are innervated by the third cranial nerve, except two, namely, the external rectus supplied by the sixth, and the superior oblique by the fourth cranial.

Muscular Imbalance.—It is due to the fact that the external and internal recti are supplied by separate nerves that imbalance occurs between these two muscles. The inadequacy of one of them, due to optical defects, is compensated for a time by excessive innervation of the other, until the overworked nerve gives way and becomes paralyzed. The sound nerve continues to innervate the opposite muscle which pulls the eye in its own direction.

Paralysis of the third nerve causes **divergent,** of the sixth **convergent, strabismus.**

Paralysis of the fourth nerve causes inability to turn the eye downward and outward.

Lucitas.—When the third nerve is paralyzed, not only dies it produce **divergent squint,** but inasmuch as all other muscles which might move the eyeball are controlled by the same nerve complete immobility of the eyeball results. This is known as **lucitas.**

Accommodation is the faculty of increasing the convexity of the lens. It is accomplished by contraction of the ciliary muscle, which draws the choroid forward, thus releasing the suspensory ligaments and allowing the lens by its elasticity to assume a more convex form. Accommodation takes place for the purpose of focussing divergent rays, i. e., for sighting near objects, and is relaxed to focus parallel rays, i. e., for sighting distant objects. The exercise of this faculty belongs to the province of optics, and will not be further discussed here.

Presbyopia.—In persons over forty-five and in younger persons under certain pathological conditions, the lens becomes hard and inelastic, and accommodation is greatly limited.

Innervation of Accommodation.—The faculty of accommodation is innervated by the third nerve, through its branches to the ciliary muscle, known as the ciliary nerve.

Paralysis of Accommodation results from (a) central lesion of the fourth ventrical, as from **brain, tumor, cerebral sclerosis, toxins (diphtheria), constitutional diseases,** (b) interruptions of the course of the third or ciliary nerve, as in **cerebral tumors, neuromata, etc.**, or (c) peripheral paralysis of the ciliary nerves, as in the action of certain drugs, notably **atropin.** The location of the lesion may be diagnosed by the involvement or otherwise of other muscles.

Spasm of Accommodation results from over-use of the ciliary nerves, and is a common accompaniment of errors of refraction.

Convergence is the faculty of directing the yellow spots of both eyes toward a point for the reception of divergent rays, i. e., toward a near point, and is accomplished by means of the internal recti.

Innervation is mediated by the third cranial nerve.

Convergence and accommodation are closely associated, and in the normal eye are performed in direct

ratio, but they are quite independent faculties and may be exercised independently.

Anomalies of Convergence have already been discussed under the heading of **ocular movements.**

The Iris serves as a cut-off to the light that enters the eye, much the same as the shutter of a camera, so that the image upon the retina may be clear. Its circular muscle (sphincter), contracting, contracts the pupil; its longitudinal muscles, by contracting, dilate the pupil.

Sluggish or Immobile Pupil results from adhesion of the iris, to the lens in **iritis** with exudation.

Photophobia and Disordered Vision are seen in **iritis** because of interference with the regulation of light entering the eye.

The Pain of iritis is spasmodic because it increases with contraction of the longitudinal muscles, and worse at night because these muscles are then in steady contraction.

Innervation of the Iris is a reflex phenomenon. The afferent stimulus is furnished by light falling upon the retina, the impulse being carried by fibres which accompany the optic nerve as far as the corpora quadrigemina, where they branch off to the centre in the fourth ventricle. The efferent (motor) impulse is conveyed to the sphincter by means of the third cranial nerve through the ciliary ganglion. The longitudinal muscles are innervated by the sym-

pathetics. It is an involuntary phenomenon, which may be inhibited, but not consciously, by the brain, as in case of emotions, nervous conditions, etc.

Mydriasis (dilation of the pupil) is caused by any condition which (a) paralyzes either the central or peripheral end of the third nerve, (b) inhibits its functionation through the brain, or (c) stimulates the sympathetic.

Tumors of the brain, and of the **third nerve,** and the action of certain drugs, notably **atropin,** are examples of the first class.

Fevers, comatose conditions, hemasthenia, tuberculosis, and **nervous depression** are instances of the second class.

Pain, sensory irritation, and **visceral diseases** furnish illustrations of the third variety.

Myosis (contraction of the pupils) on the other hand, results from influences which (a) irritate the third nerve, or (b) depress the sympathetic.

Photophobia, from any cause, **brain tumors, meningitis** and certain drugs, as **eserin,** are instances of the former; **spinal sclerosis, aneurisms** (by pressure) and **opium** of the latter.

Argyll-Robertson Pupil, in which the pupil reflex is wanting, is seen in **tabes dorsalis** and **dementia paralytica,** supposed to be due to involvement of Minert's fibres.

Wernicke Pupillary Reaction is a test for determining the location of an injury to the optic tract, (usually employed in cases of hemianopsia). If the

reflex is present, in the half of the eye that is affected, then the lesion is back of the corpora quadrigemnia; if not, it is in front of these ganglia; because at that point the reflex path leaves the optic tract to go to the ventricle.

Hippus, a periodic contraction and dilatation of the pupil, due to alternate excitation and inhibition, is seen in disorders affecting the emotions, as in **mania, hysteria,** etc.

* * *

The Humors of the eye, consisting of the aqueous, the vitreous, and the lens, serve as a refracting medium for rays of light, their resultant index of refraction, together with that of the surfaces of the cornea, lens, and vitreous, being just sufficient in a normal eye to focus parallel rays on the retina.

Inter-Ocular Tension results from a disturbance between the secretion and absorption of the humors. This condition is known as **Glaucoma.**

The steady increase of internal pressure in this disease produces a characteristic train of symptoms, such as steady pain, cupping of the disc, vascular stasis, dimness of vision, and, if not relieved, eventual blindness due to destruction of the eye.

Scotoma (blind spot) is frequently due to **opacities in the humor,** especially the vitreous, in which case they are seen by the patient as spots before the eyes (muscae volitantes).

Cataract, an **opacity of the lens,** produces, of course, diminished acuteness of vision, most marked

when it is central, least marked when it is peripheral in location. Vision is better in dim light, because the pupil is then dilated and more rays (relatively) enter the eye.

Increase in index of refraction, due to increased density of the lens, occurs in **cataract** producing **myopia** (short sight).

Stimulation of the Retina.—The light, falling on the retina, stimulates the rods and cones, which in turn, transmit the vibrations to the fibrils of the optic nerve. Inasmuch as the image on the retina is a crossed one, the halves of the retina receive stimuli from the opposite field of vision, i. e., the nasal half receives light from the temporal field and the temporal half from the nasal field. The character of the impulse generated by the light is not clear, some holding it to be thermic, some electric, some chemical. Certain it is, however, that the rods and cones transform the light vibrations into another type of impulse, for light falling directly on the optic nerve makes no impression (blind spot).

In the ordinary use of the eye, the visional perception is concentrated entirely upon those stimulations which fall upon the central portion of the retinal field; those which fall upon the peripheral areas make no conscious effect upon the brain unless special conscious effort is made to perceive them.

Hemianopsia (blindness of one-half of the retina) must be subjectively diagnosed with a view to the

crossed rays—paralysis of the nasal half producing blindness to objects in the temporal field of vision, and vice versa.

Flashes of Light are seen in **retinitis,** due to the sensitive condition of the retina to stimulation, but steady over-stimulation is almost unknown, because the inflammation quickly renders the rods and cones impermeable to stimulus.

Diminution of Vision is far the commoner symptom in all forms of **retinitis,** also in **degeneration of the retina, glioma** (tumor of the retina), **anemia,** due to poor circulation.

Sudden Blindness results from embolism of the **central retinal artery,** cutting off its entire blood supply and **gradual blindness** from **retinal thrombosis.**

Scotoma (blind spot) results from a defect in the retina. If the defect is in the peripheral field the spot is not perceived by the patient; but when it is in the central field the patient perceives it as a fixed defect of vision.

* * *

The Optic Nerve serves to conduct the light impulses from the retina to the chiasm, each optic nerve conveying impulses from its respective retina alone.

Lesions of the Optic Nerve alone, naturally affect only that eye supplied by the pathologic nerve. Inasmuch, however, as **optic neuritis** (practically the only known pathologic condition) is usually due either to brain lesions or to infectious diseases, it is

usually bilateral. It is, of course, always attended with diminution of vision, and eventually, if not cured, by blindness.

Complete Blindness of one eye always indicates injury to the optic nerve for reasons explained under The Optic Tracts.

* * *

The Optic Tract.—At the chiasm the impulses decussate, those from the right half of each retina proceeding to the right occipital lobe, and those from the left half to the left. Impulses from the macula lutea, however, go to both lobes. Hence, remembering the crossed image on the retina, visual impulses excited by objects in the left visual field are represented in the right occipital hemisphere, and vice versa.

Hemianopsia.—This arrangement makes possible that frequent form of visual disturbance known as **hemianopsia** or **hemianopia**, paralysis of one lateral half of the retinal field, manifested, of course, by blindness to the opposite visual field.

Temporal Hemianopsia, i. e., blindness to both temporal fields, must necessarily be due to an interruption involving the inner half of the optic tracts, viz., either one lesion in the center of the chiasm, or two separate lesions, one in each occipital lobe.

Nasal Hemianopsia, i. e., blindness to both nasal fields, is due to interruption of the outer halves of the tract, and must be two separate lesions, one each side of the chiasm, or one in each occipital lobe.

Homogeneous Hemianopsia, i. e., blindness to the nasal field in one eye, and to the temporal field in the other, is due to an interruption of one whole tract, or destruction of one whole occipital lobe.

Scintillating Scotoma, temporary hemianopsia, due to circulatory disturbances in one of the occipital lobes is seen in **migraine.**

Complete Blindness of One Eye, as will readily be seen, must be due in every case to a lesion in front of the chiasm, as only there are both fields of one eye alone represented.

Visual Centre.—From both the occipital lobes the impulses are conveyed to the visual centre in the left frontal convolution of the cerebral cortex, where all the areas of both eyes have central representation.

Functional Disturbances of the centre have already been fully discussed under the Nervous System, q. v.

Congenital Amblyopia, a defect of vision, not remediable by glasses, and usually associated with high degrees of refractional error, is due to the fact that a distinct image never having been thrown upon the retina, the brain has never learned to interpret the stimulus aright.

Theories of Color Perception.—Two theories prevail to account for color perception.

The Young-Helmholz Theory holds that there are present in the normal retina three photo-chemical

substances, whose decomposition imparts to the optic nerve three distinct sensations. The maximum stimulation of these separate substances is produced respectively by red, green and violet waves, but every spectrum wave stimulates all three to a greater or less degree, and the relative degrees of stimulation thus produced in the three substances determine the color sensation perceived by the brain.

The Hering Theory also assumes the existence of three photo-chemical substances in the retina, but attributes to each a double stimulative faculty, certain spectrum waves causing assimilation of the substance and other waves disassimilation, these two effects in their turn producing correspondingly different color sensations. The double capacity and stimulation effect of these substances may be thus expressed.

	Assimilation.	**Disassimilation.**
Red-Green	Red	Green
Yellow-Blue	Yellow	Blue
White-Black	White	Black

Varying degrees of assimilation and disassimilation in the various substances, of course, produce corresponding combined color sensations.

Color Blindness.—Frequently a patient is met with who is unable to perceive certain colors, and is therefore said to be **color blind** to that particular color and its combinations. The commonest colors to which color blindness is found are **red** and **green.** This condition may be (a) **retinal,** i. e., due to a de-

fect in the retina, by far the most common variety, or (b) central, i. e., due to a defect in the visual center. Whichever of the two theories above outlined be accepted, the retinal defect must consist in an absence of the photo-chemical substance whose maximum modification is brought about by the spectrum wave corresponding to the color to which sensation is lacking.

Because of its investigation by Dalton, this condition is sometimes known as Daltonism.

HEARING.

The Muscular Movements of the ear are limited to the action of the tensor tympani and stapedius, both of which render the tympanic membrane more receptive to sound, the former by direct tension of the membrane, the latter by traction on the stapes.

Innervation of these muscles is mediated by the fifth cranial nerve.

In Facial Paralysis (of the fifth nerve) the faculty of rendering the ear drum tense is lost, and the hearing is impaired to that extent.

The Tympanic Membrane is set in motion by the air waves, and by virtue of its funnel-like structure, transmits them in diminished amplitude and increased intensity to the ossicles.

Imperfect Vibration of the tympanic membrane occurs in any condition which (1) interferes with the diaphragmatic adjustment of the membrane, as in **myringitis** (inflammation of the drum), **chalk deposits** on the drum, **catarrh,** and **impaction of cerumen,** all of which thicken the membrane, and in **perforation,** which destroys its tension and integrity; and (2) bars the meatus leading to the drum, as in **catarrh, impacted cerumen,** and **growths of the meatus.**

The Eustachean Tube admits air to the tympanic chamber, thus maintaining equal atmospheric pres-

sure on both sides of the membrane and enhancing its delicacy of vibration.

Bulging of the Membrane, and consequent impairment of its vibrations, result from blocking of the tube, as in **exudative inflammation, catarrh,** and **abscess.**

* * *

The Ossicles, comprising the malleus, incus and stapes, receive the vibrations of the tympanic membrane and transmit them to the membrane of the fenestra ovalis. By virtue of their arrangement they also diminish the amplitude of the vibrations (Helmholz estimates by one-third) and increase their intensity (according to the same authority, by one-half).

Impaired Hearing results from any influence which either changes the relative sizes and relations of the ossicles as **hypertrophy** due to catarrh, **caries,** etc., or interferes with their free motion, as **ankylosis.**

* * *

The Perilymph, filling the internal ear, receives the vibrations from the fenestra ovalis, and transmits them to the rods of Corti in the cochlea.

Rinne's Test for determining the location of a lesion causing deafness depends upon the fact that if the course from the external meatus to the fenestra ovalis is interrupted while the internal ear and auditory nerve are intact, vibrations will be com-

municated to the perilymp more readily by way of the bones of the head than by the meatus (Rinne +), the lesion is located in the external or middle ear; but if the contrary (Rinne —) the lesion is in the internal ear.

Weber's Test shows that in affections of the middle ear and integrity of the nervous mechanism, a tuning fork held up the vertex is heard better in the deafer ear (Weber +) and vice versa (Weber —). This depends upon the fact that where the middle ear is blocked the vibrations received through the head bones find no outlet through the meatus and are confined to the perilymph, being thereby intensified.

The Rods of Corti, comprising the organ of Corti, perform the essential part of the function of hearing. They are from 16,000 to 20,000 in number, and are stimulated by sound waves in much the same way that the rods and cones of Jacob's are stimulated by light waves.

The Piano Theory of Hearing assumes that each rod of Corti is a resonator, responding to a distinct pitch or wave-length, and that simple and compound sensation of sound depend upon the stimulation of one or more of these resonators, recording a specific reaction in the brain for each rod.

The Telephone Theory holds that the compound sound wave stimulates the organ of Corti as a unit, and that the analysis of the sensation is performed in the brain centre.

In Disease of the Labyrinth extending from the middle ear, the hearing for high pitched notes is greatly impaired, because the few short rods at the commencement of Corti's organ, hear the vestibule resonate to high pitched sound waves.

Auditory Limits.—The range of perceptible sound waves varies in different individuals, but the average audible gamut is from 30 vibrations per second (low pitch) to 40,000 per second (high pitch). Slower vibrations than the former, if perceived at all, are usually only perceived as stimulations of the sensory nerve of the tympanum—the auditory nerve does not react to them; or else the auditory nerve responds to their overtones, for particulars of which a work on acoustics must be consulted.

Pathological Limitations of Pitch are caused by **ankylosis** of the ossicles which prevents those bones from vibrating rapidly.

The Auditory Nerve collects the vibrations from the rods of Corti and transmits them to the auditory centre in the temporal lobe, each ear being separately represented in each corresponding temporal lobe.

Sudden Deafness is a prominent symptom of Meniere's disease, caused by a hemorrhage into the auditory nerve or labyrinth.

Timitus is a symptom of all ear disorders which increase tension in or shut off egress from the internal ear, because the physiological noises, such as

circulation, muscle tonus, etc., which under ordinary conditions are too diffused to be heard, are confined to the labyrinth and become audible.

Vertigo and Dizziness accompany all such conditions because of the interference with the integrity of the semi-circular canals and consequent disturbance of co-ordination. (See **Nervous System.**)

Auditory Centre.—The auditory centres in the temporal lobes transmit the sensation of sound to the auditory centre in the left frontal.

Affections of the Auditory Centre are fully dealt with under **Cerebrum.**

SMELL.

The Olfactory Mechanism consists in the contact of chemical particles of the odoriferous substance, usually but not necessarily in gaseous forms, with the upper roof of the nasal chamber where they stimulate the outspread filaments of the olfactory nerve. This contact may be effected either by way of the anterior or the posterior nares. In the latter case it enters largely into what is commonly regarded as taste.

The olfactory faculty is probably the most acute and delicate of the senses, no adequate measurement of the threshold stimulus having yet been found.

Anosmia (loss of smell) is, of course, seen in all disorders which (a) impair or destroy the mucous membrane in which the filaments are spread, as in **rhinitis,** especially the chronic hypertrophic and atrophic forms, **catarrh,** etc., or (b) obstruct the nares, as in **adenoids, polypi, hypertrophy of the turbinate,** new growths and allied troubles.

Hyperosmia (increase sensitiveness to smell) and **Parosmia** (perverted sense of smell) are usually nervous disorders (vide infra).

The Innervation of Smell, so far as we know it, is a simple matter. The stimulation of the filaments promulgates a nerve current along the olfactory nerve, which reaches the olfactory centre in the

frontal lobe and registers itself there as a sensation. It is held that the various qualities of odor are due to stimulation of different fibres of the nerve.

Anosmia results from any condition which (a) destroys or impairs the path of innervation, or (b) renders the nerve or centre irresponsive to stimuli. **Brain tumors** and **cerebral softening** are the most conspicuous examples of the first; **hysteria, melancholia** and **nervous depression** of the second.

Hyperosmia is usually an accompaniment of hysteria, which renders the olfactory center unusually acute.

Parosmia is also the result of such functional psychic aberrations as **hysteria, neurasthenia,** and **melancholia,** but may be due to congenital absence of certain specific fibres in the nerve.

TASTE.

The Mechanism of Taste consists in the contact of the sapid substance, in solution, with the surface of the tongue, where it stimulates the filaments of the nerves of taste. It is absolutely essential that the substance be in solution; taste is impossible on a dry tongue.

Absence or Diminution of Taste results from any condition which destroys or impairs the mucous membrane in which the nerve filaments or taste buds lie, as **cancer, ulceration, tuberculosis,** and severe forms of **glossitis.**

N. B. Absence of taste, so called, in catarrh, is due to impairment of retronasal olfaction.

Perversion of Taste (far more frequent than diminution) may arise from conditions of the tongue and palate which alter the sapid substance, as **salivary disorders (mumps), digestive troubles** (from food coating), **fevers** and **sore throat** (from epithelial coating), etc. The anomaly is therefore not really a perversion of the sense, but of the sapid substance.

* * *

Classification and Distribution of Taste Sensations.—There are but four fundamental taste sensations, of which all others are combinations and modifications, and while these are distributed over the tongue and palate differently in different individuals, yet

the general average preponderance of the various sensations is as follows:

Sweet.—Tip and forepart of tongue.

Bitter.—Back of tongue.

Acid.

Salty.

Dissociated Taste Sensations are diagnostic of disorders of the tongue occurring in patches, which can be located (but not with exactness) by the dominant sensations.

* * *

Innervation of Taste is mediated by the lingual (a branch of the inferior maxillary division of the fifth) nerve, supplying the anterior two-thirds of the tongue; and the glossopharyngeal, supplying the posterior third. Recent observations by Cushing of Baltimore demonstrate that that the former fibres do not accompany the fifth nerve through the Gasserian ganglion, but pass to the Petrosal ganglion and thence to the fourth ventricle. The same evidence indicates that the latter fibres pass directly to the gessiculate ganglion and enter the brain with the seventh nerve.

The Sensation does not depend upon any specific character of peripheral stimulation but upon the specific reaction of the cerebral termini.

Absence of Taste results from any condition which interrupts the course of the nerve currents con-

cerned, or the recording of those impulses in the brain, as **brain tumors, neuromata, neuritis,** etc.

Hypersensitive Taste is seen in conditions which exalt the nervous functions, as **hysteria,** and the influence of certain drugs, **strychnia,** etc.

Perversion of Taste is almost always due to perversion of psychic function, as in **melancholia, hysteria, neurasthenia, hypochondria,** etc.

REPRODUCTION.
OVULATION.

The Testicle is the essential organ of ovulation in the male. In the seminiferous tubules of these glands the spermatogenic cells (a very elementary variety of epithelial cells), by a process of Karyokinesis, evolve the spermatozoa. These are produced in enormous numbers, from twenty-five to a hundred million of them being contained in every cubic centimeter of ejaculated semen. The secretion of these cells begins at the age of puberty, and continues indefinitely, doubtless under the nervous influences of the sympathetics.

Azoospermatism, an absence of spermatozoa in the semen, results from secretory disablement of the testicles. Absolute azoosperma is a rare condition, requiring complete disablement of both testes, but comparative azoosperma is not infrequent, consisting of a diminution in number or activity of spermatozoa. It is seen in malignancy of the testicle, orchitis, syphilis, tuberculosis, and sexual exhaustion from excessive coitus. Its effect is of course total or relative sterility.

The Vas Deferens carries the spermatozoa from the testicles as fast as they are produced, to the seminal vescicles.

Azoospermatism is sometimes seen in conditions which impair the potulency of the vasa deferentia,

preventing the passage of the spermatozoa into the vescicles.

The Seminal Vescicles fulfill two offices. They serve as reservoirs for the spermatozoa, and their mucous lining secretes a fluid which is mixed with the spermatozoa. The specific influence of this secretion is unknown, but it serves at all events to increase the motility of the spermatozoa.

The Prostate and Cowper's Gland also contribute to the constituency of the semen. The secretion of the former, which consists chiefly of sodium chlorid, dilutes the semen and decreases its consistency, thus rendering it very motile.

In Seminal Vesciculitis, especially of the chronic type, the walls of the vescicles frequently become adherent, and the spermatozoa are unable to pass out into the semen. Such a condition of course produces asperma and sterility.

Prostatitis, especially when chronic, often results in sterility, due to the absence of prostate fluid, and consequent immotility of the spermatozoa. This condition of the semen is known as colloid semen.

The Semen is a mixed product, consisting of secretions of the testes, vasa deferentia, vesiciculae, seminates, prostate, and Cowper's gland. It is a whitish viscid fluid, alkaline in reaction, specific gravity of about ——, and having a characteristic odor. It contains water and sodium chlorid (the

greater bulk) nuclein, protanin, proteid, lecithin, cholesterin, fat, and inorganic salts.

The Ovary is the essential female organ of ovulation. In it the ovum is developed, from the Graafian follicle. As the ovum grows the follicle is forced to the surface and the circulation at the point of tension is cut off, producing necrotic changes, until the follicle finally bursts and liberates the ovum. The cavity thus left is filled with a yellow filtration liquid and is known as the corpus luteum. The life of the corpus luteum is about three weeks, except when the liberated ovum is fertilized, under which circumstance it is not absorbed for several months (corpus luteum of pregnancy).

Like the ovulation of the male, ovarian activity begins with puberty and lasts until forty or forty-five years of age, at which time the sexual life of the female ends.

Absolute Sterility, such as occurs in the male in the absence of the testical secretion, occurs in the female in total disability of both ovaries. Fortunately such a condition is exceedingly rare, and is only found in such uncommon cases as **bilateral ovarian cysts, ovarian atrophy** and **profound constitutional diseases** in which the ovarian vascularity suffers in common with the other organs.

As in the case of the male, however, sterility from complete suppression of ovulation is extremely rare.

Excessive Ovulation results from a hyperemic

state of the ovary, such as is induced by **frequent coitus, ovaritis,** and any form of **pelvic congestion.** In such cases the vitality of the rapidly matured ovum is below par, so that the net result of such conditions is diminished fertility, or relative sterility.

Premature Menopause is induced by those conditions which unduly increase ovulation, owing to the rapid exhaustion of ovarian vitality.

The Fallopian Tubes receive the discharged ovum from the ovary, and convey it by ciliary motion, in a varying length of time, to the uterus. Evidence tends to show that the ovum is not necessarily discharged into the tube on the same side as the functionating ovary, but is discharged into the pelvic cavity and caught by the fimbriated extremities of either tube, or may never reach the tube at all but be disintegrated and absorbed in the abdominal cavity.

Sterility is more frequently due to disease of the tubes than to all other causes combined. Any condition which impairs the integrity of the tubes naturally hinders the passage of the ovum and prevents its meeting with the male element. Conspicuous examples are seen in **salpingitis, hydro-** and **pyo-salpinx,** and **tubercular infiltrations of the tubes.**

Ectopic Gestation of the abdominal type occasionally occurs as the result of the failure of the ovum to reach the tube, and the migration of a male sperm-cell into the abdominal cavity, where the two meet and fuse.

Menstruation is a periodic phenomenon in the female process of ovulation, in which the ovaries, fallopian tubes, and uterus take an active part. There can be little doubt that menstruation bears a sequential relation to the liberation of the ovum, the general idea being that the growth of the Graafian follicle furnishes in some way, a stimulus to increased uterive metabolism.

The Process of Menstruation is divided by Howell into four stages, as follows: (1) Period of growth, five days, characterized by a rapid increase in the uterine stroma, blood vessels, epithelium, etc.; (2) period of degeneration, four days, during which capillary hemorrhage takes place and the epithelium is degenerated and cast off; (3) period of regeneration, seven days, during which the mucous membrane returns to its normal condition; (4) period of rest, twelve days, during which the endometrumi remains quiescent.

The Rationale of Menstruation is undoubtedly that of a preparation of the uterus for the reception of the ovum, whether we adopt the view that the congestion of the membrane constitutes the preparation or the raw surface left by degeneration.

Amenorrhea is commonly understood to signify simply a failure of the visible flow in the second period of menstruation, and as such may result from obstructive and anatomic causes. True functional amenorrhea, however, is a failure of the whole proc-

ess, and usually depends upon (a) some constitutional dyscrasia by which vascularity of the generative organs suffers with that of the other organs, or (b) some local vaso-motor disturbance in the generative tract.

Of the former variety, **anemia, tuberculosis,** and **neurasthenia** are frequent examples; of the latter type, **trauma, surgical shock, cold,** etc.

Menorrhagia (excessive flow), on the other hand, results from those conditions which increase uterine congestion and metabolism, either systematically, as in fevers, or locally, as in **metritis, local malignancy,** and all forms of **pelvic inflammation.**

Dysmenorrhea, outside of those cases due to anatomic anomalies, is comparatively rare. True functional dysmenorrhea is usually due to a neurosis of some kind, as **neuralgia, neurasthenia,** or **hysteria.**

Suspended Menstruation With No Other Signs of Disturbed Health, especially in a woman who has heretofore been regular, is almost infallibly indicative of pregnancy.

Vicarious Menstruation.—Sometimes the mucous membrane of the uterus fails to undergo degeneration, and under the increased vascular tension the capillaries in other parts of the body, such as the breasts, stomach, lungs, nose, etc., breaks down in hemorrhage.

Supplementary Menstruation occurs when the capillaries of other organs break down in addition to uterine disintegration.

FERTILIZATION.

Erection is an important, though not absolutely essential, factor in fertilization because it facilitates the entrance of the penis into the female vagina, and the depositing there of the spermatozoa. It is accomplished by means of engorgement of the vessels of the cavernous spaces of the erectile tissue, whereby the penis is enlarged and rendered hard and erect.

Innervation of Erection is mediated by the nervi erigentes, composed of sympathetic fibres from the sacral segments of the cord by way of the pelvic plexus. The act is a reflex one, whose stimulus may arise from the brain, as in the case of erotic thoughts, or from irritation of the sensory nerves of the testes, urethra, or glans penis, and is effected through a centre in the lumbar cord. Experiment shows that the nature of the efferent impulse is a vaso-dilator influence, but its precise rationale is obscure.

Impotence, as to erection, results from any condition which (a) interrupts the course of the reflex, or (b) inhibits it from the higher centres.

The former conditions are found in all of those spinal diseases which impair the integrity of the lumbar centre, as **tabes dorsalis, sclerosis myelitis**, and in all of those nervous diseases in which the general reaction to stimulus is lowered, as **neurasthenia, tuberculosis, diabetes**, etc.

The latter type of impotence is generally known as psychical impotence, and is seen in **hysteria, melancholia,** and **neuroses** of all kinds.

Priapism, on the other hand, arises from any state which (a) renders the reflex abnormally sensitive, or (b) stimulates the cerebral end of the tract. In the former class are **inflammatory spinal diseases,** as early **myelitis, meningitis, spinal** and **cerebral hyperemia,** and **growths.** In the latter class are **manias, epilepsies, hysteria,** etc.

Ejaculation is accomplished by a vigorous and sudden contraction of the muscles of the vaso deferentia, seminal vescicles, perineum, and urethra, in the sequence indicated, throwing the semen into the female vagina. It is the last part played by the male in the process of reproduction.

Innervation of Ejaculation is similar to that of erection, is effected through the same spinal centre, and stimulated by an intensification of the same peripheral stimuli. It is, however, rarely precipitated by direct cerebral stimulation, as erection is.

Impotence, as to ejaculation, depends in a general way, upon essentially the same functional disturbances as failure of erection, and the classification under that head may be accepted as applying to this process. It may be said, however, that this faculty fails earlier than that of erection; and to other causes must of course be added those conditions in which no semen is secreted. The latter, however, do

not of themselves really influence the functional performance of ejaculation, as the reflex takes place even though no fluid is ejaculated.

Premature Ejaculation and Emissions occur under the same conditions as those enumerated under priapism, q. v.

* * *

Impregnation of the ovum by the spermatozoon takes place usually in the Fallopian tube. The probability is that the ovum exerts a chemotaxic attraction for the spermatozoon within certain limits of distance. The minute nature of the impregnating process belongs to the science of embryology, and will not be discussed here.

Infecundity, due to impaired integrity of the tubes, is, as already stated, far more frequent than from any cause. Any condition which renders the tube impassable and prevents the passage of the male spermatozoon is fatal of course to impregnation. Such conditions are found in the **plastic adhesions** caused by **salpingitis** (especially gonorrheal), **tubal tuberculosis,** and in **pyo-, hydro-,** and **hematosalpinx.**

Tubal Pregnancy.—In some instances the impairment of the tubes, although not sufficient to hinder the passage of the spermatozoon, is enough to prevent the fertilized ovum from passing down into the uterus, in which case it remains in the tube and there develops into a fetus. This is known as **tubal ectopic pregnancy.**

GESTATION.

The Uterus is the essential organ of gestation. The fertilized ovum attaches itself to the decidual membrane furnished by the menstrual process, and develops into the fetus. Thereafter the menstrual cycle is suspended until gestation is completed and the uterus emptied of its contents.

The uterus enlarges with the growth of the fetus, and at about four months of pregnancy, becoming too large for continence by the pelvis, it rises into the abdominal cavity.

Incapacity for Gestation may arise from any condition of the uterus which renders it unfit for the attachment of the placenta. Such incapacity usually results from either an anemic and poorly nourished state of the uterus, or in **constitutional diseases, malignancy, anemia,** or from the opposite condition of **hyperemia,** as in **metritis, endometritis, pelvic congestion, tumors,** etc.

Abortion due to the premature severing of the ovum from the uterine wall often results from the same causes as those just enumerated and for the same reason.

The Placenta constitutes the vascular medium through which nutritive material passes from the maternal blood to the fetal, and waste products from the fetal to the maternal, by the agency partly of

osmosis and diffusion, but chiefly, no doubt, of the epithelial cells of the villi, of the placenta. The maternal and fetal blood do not come into actual contact.

The metabolic exchanges thus effected include practically the same as those of the independent organism, namely, proteids, carbohydrates, and oxygen on the one hand, and nitrogenous wastes and carbon dioxid on the other.

Vomiting of Pregnancy.—In the early weeks of gestation the volume of maternal blood has not yet adjusted itself to the increased demands upon it. The consequence is an impoverished and toxic condition of the maternal blood which poisons and irritates the nerve centres and causes nausea and vomiting. "Morning Sickness" is the expression of a cerebral anemia due to assuming the vertical posture after sleep.

Death of the Fetus of course follows interruption of the communication between the maternal and the fetal organism—in other words, any impairment of the integrity of the placenta.

Abortion, in the sense of premature termination of gestation, occurs more frequently from placental impairment than from any other cause outside of mechanical trauma.

Placental Impairment.—As the placenta is essentially a vascular organ it is subject to all of the disorders that affect other vascular organs. Such dis-

orders may be (a) of a constitutional nature, affecting the placenta in common with the rest of the organism, or (b) local in character, peculiar to the placenta. Of the former **syphilis** is by far the most common offender; **tuberculosis, malignancy, diabetes, nephritis, toxemia** (of any kind), all of which degenerate the placental tissues; in the latter variety are included **embolism, thrombosis,** and **apoplexy,** of the placenta, **hydatid pregnancy, infarctions, calcareous** and **fatty degenerations,** etc.

Infection may pass in either direction, from mother to fetus or from fetus to mother, through the placental medium. In this way **syphilis, tuberculosis** and other infectious diseases are frequently transmitted.

Malnutrition.—Those placental conditions cited as causes of fetal death commonly are not sufficient to kill the fetus but result in its insufficient or vitiated nutrition.

* * *

The Mammary Glands during gestation exhibit considerable growth and activity in their true glandular structure, and in the latter part of pregnancy secrete a scanty fluid known as colostrum.

PARTURITION.

Muscular Contractions of the uterus occur spontaneously about 280 days after conception, and continue periodically at varying intervals of twenty minutes at the beginning to one minute at the last, until the fetus is expelled. The contractions are confined to the upper part of the uterus, the lower walls offering no resistance to the expulsive distension; a detailed account of the process of parturition belongs to a study of obstetrics, and will not be considered here.

Protracted and Precipitated Labor are the two opposite deflections from the normal process, depending upon a disturbance of the normal balance between the uterine forces and the dimensions of the fetus.

Protracted Labor is due either to a subnormal character of uterine contractions, or to a super-normal size of the fetus. The former is known as **uterine inertia,** and is seen in all conditions of poor constitutional health, and in **myoma** of the uterine walls, due to **tumors, fatty degeneration, anemia, tuberculosis,** etc.

Precipitate Labor results from either abnormally powerful uterine contractions or an abnormally small fetus, usually the latter.

Obstructive and anatomic causes of dystocia are

not functional in their nature and will not be discussed here.

* * *

Innervation of Parturition is a matter of some obscurity. Undoubtedly it is a reflex phenomenon whose afferent stimulus is furnished in some way by the resultant conditions attendant upon a matured fetus, but the precise modus operandi of this stimulus is unknown. Experiment shows that the reflex is independent of the cerebro-spinal centres, although it is well known that the process is influenced, and may even be inaugurated, through these centres.

Premature Parturition (Abortion) results, outside of trauma and physical accidents, from any condition which sets in motion the stimulus above referred to before the fetus is matured. Until more is definitely known of the nature of that stimulus, little can be predicated about its premature excitation.

Accepting the theory that the normally matured fetus acts as an independent foreign body in the uterus, then any condition which renders the fetus an independent foreign body—in other words, anything which terminates the physiological union of mother and fetus causes expulsion. Among such conditions are **constitutional disease,** causing placental degeneration, **hyperemia of the pelvis** from exercise or coitus, **placental apoplexy.**

Mental Shock may also start premature contractions through the cerebro-spinal tract.

LACTATION.

The Breasts, immediately after delivery, secrete an abundant supply of milk, and continue to do so, under normal conditions and within certain limits, as long as the child is kept at the breast.

Innervation of Lactation, like that of parturition, is obscure. The breasts are physiologically connected with the uterus and ovaries, and undoubtedly receive specific nerve stimuli both from the process of ovulation and parturition, but the precise mechanism of these stimuli is at present unknown. Like the process of parturition, lactation is directly influenced through the cerebro-spinal centres.

Agalactia, absence of milk, in its complete form, is extremely rare, but relative agalactia is not uncommon, depending, of course, on either a disability of the glands themselves, as in **malignancy, anemia** (lack of vascular nourishment), **diabetes, tuberculosis, syphilis** (lack of cellular nutriment) and **mastitis**; or to impairment of the nervous mechanism, as in anomalies of the uterus and ovaries (perverted stimulus), and more frequently in mental disturbances such as **melancholia, neurasthenia, hysteria, shock.**

Polylactia, an excess of milk, is very common, and results from any condition which (a) increases the flow of blood to the breasts, or (b) overstimulates the nervous mechanism. In the former class

are **mastitis, vaso-motor disturbances,** etc., in the latter **metritis, ovaritis** and **psychic influences.**

Premature Lactation is precipitated by a premature consummation of the physiological conditions which normally mature at parturition. It usually indicates premature separation of the fetus from physiological dependence upon the mother, hence fetal death.

* * *

The Milk has an average composition as follows: Proteids, 1 to 2 per cent; fats, 3 to 4 per cent; sugar, 6 to 7 per cent; salts, .01 to .02 per cent. There are also negligible quantities of nitrogen, urea, creatin, lecithin, cholesterin, etc. The bulk of the proteids are in the form of casein. This and the milk sugar are true secretory products of the mammary glands, as neither preexists in the maternal blood.

Infant Feeding must be based upon the normal composition of human milk, as that must be regarded as the ideal supply of the infant's nutritional needs.

Abnormal Proportions of the constituents of the milk occurs in cases where the maternal metabolism exhibits corresponding inequalities. Thus when proteid metabloism predominates the proteids in the milk are high, and vice versa.

Abnormal Proportions of the constituents of the milk are seen in cases where the maternal metabolic balance exhibits corresponding inequalities. Thus,

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